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GLASGOW HOSPITAL REPORTS

EDITED FOR THE COMMITTEE

BY

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AND

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VOL. II.

WITH FIFTY-FIVE ILLUSTRATIONS

GLASGOW
JAMES MACLEHOSE AND SONS

Publishers to the University

1900

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CASE OF A SMALL ANEURYSM OF THE FIRST
PART OF THE ARCH OF THE AORTA, OPENING
INTO THE PULMONARY ARTERY AND CONUS
ARTERIOSUS OF THE RIGHT VENTRICLE;
WITH REMARKS ON THE GENERAL SUBJECT.

By SIR WILLIAM T. GAIRDNER, M.D., K.C.B., F.R.S.,
Physician to the Western Infirmary, Glasgow.

IN the last published volume (VI.) of the *Transactions of the Glasgow Clinical and Pathological Society*, under date 10th May, 1897,¹ I brought before the Society a collection of specimens from the Glasgow Museums, including all which I found there available as “illustrating aneurysms of the arch of the aorta which either opened into, or pressed upon, the vena cava superior, or the pulmonary artery.” The collection amounted to twelve cases in all (not all strictly conformable to the title), and it left upon the mind the general impression that such cases are not only rare, but that even when thus collocated they do not contribute very much, in most instances, towards the precise clinical history and diagnosis of such lesions; except indeed in one case which I myself communicated to the *Lancet* of 22nd June, 1889, p. 1233, where the opening was into the superior vena cava, with well marked cyanosis and dropsy, and with dilated veins generally limited to the upper part of the body. The diagnosis of aneurysm in this case was not very difficult, and there was even a fact in the history which may have corresponded with the period of the rupture into the vein—viz., a “feeling as if something had

¹ See also *Glasgow Medical Journal*, 1897, Vol. II., p. 120.

given way on the left side, rather to the left of the cardiac apex" (and therefore not precisely in the situation of the actual rupture); the feeling, however, was not one of local pain, but attended by faintness and cold sweat, succeeded almost immediately by the swelling and the other phenomena observed. There were in this case loud murmurs (V.S. and V.D. in rhythm) heard over the whole front, but with mainly the distribution of the aortic double murmur. No considerable doubt was in this case entertained of the existence of an aneurysm involving the vena cava, the diagnosis being afterwards verified by post-mortem examination. The clinical facts in this case were very completely, though concisely, stated in the article alluded to; but it is obvious that when an opening takes place into the pulmonary artery or right ventricle, the localization of the anasarca as above described, is not to be expected, and the diagnosis is therefore, or at least may be, much more difficult. [See paragraph appended to this article, page 17.] The following case, therefore, having been most carefully recorded with a view to clinical teaching, and having been under observation for a sufficient time to be repeatedly observed and considered with a clinical class, appears to be a fitting contribution to the Glasgow Hospital Reports. I am greatly obliged to my friend and old pupil, Dr. Alexander Macphail, for his admirable drawings of the preparation now in the museum of the Western Infirmary, which will allow all the facts that can be brought under the eye of the reader to be vividly presented, in connection with the clinical history of the case. The report in the journal, however (Ward L., A., p. 213), is so lengthy that it will be necessary to resort to a summary which, it may be said, is *verbatim* the one that was recorded and placed before the clinical class, soon after the admission of the patient, in order to facilitate remarks at lecture, and to guide as accurately as possible the conceptions of the members of the class as to the presumptive diagnosis.

"Wm. G. L., aet. 35, engineer; admitted 16th March, 1899. Cardiac lesion of remarkably obscure origin, possibly of about twelve months' standing on admission; but, if so, characterized *only* by a certain amount of breathlessness and dis-

comfort on unusual exertion, for the greater part of that time. No dropsy, until less than a week before admission. No local pain, or other definite subjective sensations of cardiac distress. No hæmoptysis (this negative fact continued so to the end). Almost no orthopnoea.

"Previous history of syphilis, ten years ago or more. No rheumatism. No probable history of strain. Family history unimportant.

"Cardiac murmurs of complicated character, at first interpreted as being at least partly aortic (V.S. and V.D.), with pulse more or less characteristic of aortic regurgitation.

"Well-marked hypertrophy, probably of both, certainly of the right ventricle, with heaving impulse and thrill to the hand; but no palpitation complained of. Dulness to percussion (of heart) $7\frac{1}{2}$ inches transverse. Epigastric pulsation.

"Question raised of aneurysm or other lesion, involving the right side of the heart as well as the aortic valves; but no positive evidence of this obtained.

"Enlargement of liver, with induration, but no marked irregularities. Other abdominal organs not apparently altered. Urine more or less albuminous throughout."

It will be observed from the above concise statement of the actual facts observed, that there was very little in the history to guide the mind to a conclusion as to the cardiac character of the case, still less as to aneurysm, or internal rupture, at least up to the time of the occurrence of general dropsy. And this peculiar insusceptibility, on the part of the patient, to impressions of cardiac suffering, was maintained throughout the treatment, almost up to the fatal issue. Again and again it fell to be remarked that even in the presence of severe and dangerous objective symptoms—including a degree of general dropsy which required repeated puncturing of the limbs and scrotum, till it was for a time marvellously relieved under *diuretin*—the mental condition of the patient was one of apparently complacent optimism, which would hardly allow of the existence, according to his own reckoning, of any gravity in the symptoms at all. Along with this very remarkable *euphoria* there was, it is

true, an equally remarkable tendency to muttering delirium, without fever or any other apparent cerebral complication, but suggestive of danger had it tended (as was regarded probable) either in the direction of coma or of exhaustion. During nearly ten weeks of observation, however, his state in this respect remained the same, noisy and apparently very restless, especially at night, but yet always able to pull himself together, as it were, when addressed, and even up to within an hour or two of his death responding to questions, as if he felt "better" or "very well," as the case might be. His pulse was always unduly rapid (98 to 116, but always regular, increasing in rapidity latterly to 138), and his respiration appeared more or less laboured, but without any marked orthopnoea, or any considerable lividity until near the end. The treatment was mainly on a diuretic plan (salines, digitalis, diuretin, spartein, caffen, etc.), and was, up to a certain point, successful, the diuretin in particular taking great effect on the quantity of the urine, and for a time on the dropsical condition. It was found impossible, however, by any combination of these remedies, to prevent recurrence of the dropsy, and ultimately drainage by Southey's tubes and by incisions had to be employed—again for a time, with apparently good results—showing a very remarkable vital resistance to what seemed, all along, a most dangerous combination of symptoms. It is not desirable, however, here to go into more details of these fluctuations in the general state of this patient. The following extracts from the reports will bear chiefly on the objective cardiac changes, and particularly the murmurs.

Owing to the question of an aneurysm being throughout present to the mind, particular attention was given to the percussion of the manubrial and extra-cardiac area, but no definitely dull space such as would indicate a sacculated or general dilatation of the arch, could at any time be detected. The cardiac dulness, on the other hand, was manifestly extended laterally, and quite as much towards the right as towards the left. A distinct systolic thrill was felt over the right ventricle, and epigastric pulsation was distinctly present.

"At the apex, a loud V.S. murmur is heard" (first report after admission), "long and blowing, and a much fainter and shorter V.D. The V.S. becomes still louder towards the sternum, and reaches maximum intensity just over the tricuspid area, where also the V.D. is very distinct. Both murmurs are audible at the aortic cartilage, the V.S. being much less intense here than at the lower division of the sternum, and scarcely at all conducted into the neck, while the V.D. attains its maximum over the second right cartilage, and is not at all heard over the cervical vessels. No capillary or venous pulse can be demonstrated."

At this time my own conviction, founding on several very careful personal observations, was that there was probably aortic valve-disease, obstructive and regurgitant, *but also, probably, something more*. The pulse was fairly characteristic of aortic regurgitation; but the peculiar distribution of the murmurs, the thrill over the tricuspid area, the epigastric pulsation and enlargement of the liver, the great amount and persistence of the dropsy, all pointed to the implication of the right ventricle to a much greater degree than is usual in aortic disease, even when of some considerable standing. In this way the question of aneurysm, though not a matter of evidence, was distinctly before the mind from the first, and was thus introduced as a speculative diagnosis into the summary above given; and, amid all the changes taking place in the general condition as above indicated, nothing in the physical signs, or the disposition of the murmurs, occurred to suggest any change in the essential lesion, until, perhaps, seven or eight weeks, or more, after admission, when a marked increase in the complexity of the murmurs began to be observed, but without any corresponding, or at least any sudden, change in the symptoms. On 7th May I endeavoured to convey this, admittedly puzzling, change, as follows: "It has seemed to Professor Gairdner in some of the more recent observations, that there is a change in the quality and even in the rhythm of the murmurs; and this at one observation suggested the possibility of some part of them (at least) being of exocardial origin (the point was, in fact, submitted to a

number of good observers). On careful observation to-day, however, it does not appear probable that this is the case; but, on the other hand, the extremely loud and all but continuous murmurs heard over the right ventricle seem to Dr. Gairdner much more striking, and also more difficult of interpretation, than what was originally observed and reported. At the right of the sternum they are not so overpoweringly loud, and are also more rhythmic, than elsewhere; and over the aortic area V.S. and V.D. murmurs, very much of the usual character in aortic disease, can still be separately distinguished; but, on the other hand, the loud, almost roaring, hollow murmurs heard midway between the left nipple and the middle line are such as to confuse the ear as to the rhythm, more especially as the sounds (apart from the murmurs) are entirely lost, and what must be presumed to be the V.S. and V.D. murmurs (see above) are here quite continuous with one another. Various hypotheses have been entertained since this patient's admission, to account not only for the peculiarities of the murmurs, but also for the obstinacy of the dropsical symptoms, and the evident great predominance of the right side of the heart in a case where the murmurs and the pulse seemed to indicate aortic disease; the question was entertained of aneurysm, possibly communicating with the right ventricle or pulmonary artery; but, on the other hand, the absence, or slight degree, of cyanosis seemed unfavourable to that view; and nothing in the percussion at the base, or the palpation in the jugular fossa corresponds with the idea of an aneurysm of the first part of the arch. Now, however, with these extremely complex murmurs heard over the right ventricle, it seems difficult to exclude some lesion of an unusual kind involving the right side of the heart; but, so far as can be observed, the pulmonary artery is not particularly involved in it, and wherever the V.S. and V.D. can be separately distinguished, the inference would rather be that they are of aortic origin."

The above note, made sixteen days before the patient's death (which occurred on May 23rd), and used for direct bedside instruction at a time when the patient was quite in a state

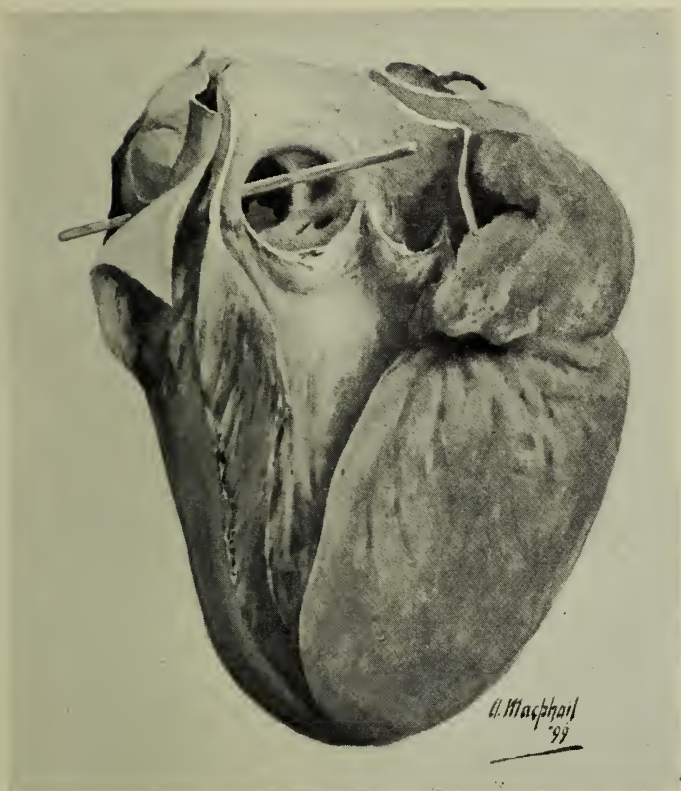


FIG. 1.--The heart and aorta laid open, showing the origin of the aneurysmal sac immediately above the anterior cusp of the sigmoid valve; the aneurysm tending towards the pulmonary artery, which is seen, laid open, to the left of the drawing, a probe being passed from the sac into the pulmonary artery by an opening which is not visible in this figure, but is prominently displayed in Fig. 2. The small opening to the right, within the sac, is that of the coronary artery.

to bear examination by a considerable number of persons, was intended specially to convey to the whole clinical class not only the absolute facts, but also the tendency of opinion on the facts, as to the changes observed in these murmurs. The diagnosis, it is true, is not perfectly accurate, inasmuch as it implies, negatively, that there was no evidence of the pulmonary artery being specially implicated, while it points, very decidedly, to the right ventricle as the seat either of rupture, or pressure by the aneurysm which, notwithstanding the absence of clear evidence by percussion, etc., had all along been regarded as not improbably existing. The presumption, to my own mind, was that a rupture directly into the pulmonary artery (or the conus arteriosus) would not only have been attended by much more considerable cyanosis (as in one or two other cases in the series above referred to), but also that in that case the murmurs would have been likely to be more strictly localized and centred, over the first and most superficial portion of the pulmonary artery itself. Nevertheless, the facts are as stated, and the difficulties here more or less successfully encountered show that the absolute diagnosis of varicose internal aneurysm, and of the seat of rupture (except perhaps in the superior cava), must still be regarded not only as among the rarities, but also among the difficult problems of physical diagnosis. In the present instance, the solution was probably as nearly attained as was possible under the circumstances.

The post-mortem examination (well illustrated as to the essential facts by Dr. Alexander Macphail's excellent sketches) gave, in summary, the following results:

"The pericardial sac contained about 6 oz. of clear serous fluid.

"The left lung was bound down by fibrous adhesions all over. The right pleural cavity contained about 30 oz. of fluid.

"*Heart.* Aortic orifice is slightly incompetent. The commencement of the aorta shows some patches of atheroma. Behind the anterior cusp of the aortic valves a comparatively wide orifice communicates with an aneurysmal sac which, passing at first to the right, bulges into the pulmonary artery.

The right anterior cusp of the pulmonic valve is stretched over the sac, and is almost obliterated. There is an aperture of communication, about $\frac{1}{8}$ inch in diameter, with the right ventricle, about half an inch below the attachment of the cusps. In the aorta, posterior to the aneurysmal opening, is another small depression, apparently a commencing aneurysm. The coronary arteries, which show some patches of atheroma, are quite uninvolved at their orifices. The mitral segments show some diffused thickening, but are otherwise normal. The pulmonic valve, with the exception noted above, is normal, as is also the tricuspid. The left ventricle measures $3\frac{3}{4}$ inch in length, and is from $\frac{3}{4}$ to $\frac{7}{8}$ inch in thickness. The right ventricle is also hypertrophied, the wall being $\frac{1}{4}$ inch in thickness. Both auricles are dilated, and contain some ante-mortem clot.

"There is venous congestion of the lungs, kidneys, liver, and spleen.

"The aorta, both thoracic and abdominal, shows numerous raised patches of atheroma, pretty equally distributed; these are soft, but show no trace of ulceration or calcification.

"The right internal jugular vein is found thrombosed, the thrombus extending down to the junction of the subclavian vein, and partly obstructing the latter. The thrombus is of firm consistence, laminated, pale, and in parts adherent to the wall." (This lesion does not appear to have been noticed during life.)

It has been already remarked that, especially in the earlier observations of the case, the radial pulses had in general the characters of those of aortic disease. The "water-hammer" character to the finger was frequently very well marked, and the sphygmographic tracings obtained (which were sometimes difficult to procure owing to the dropsy) were quite in conformity with this view, although under the influence of digitalis, etc., they varied considerably as to the extent of the primary wave and the distinctness of the dicrotic and other waves. In one tracing, obtained on 12th May (eleven days before death), the tracing was decidedly hyperdicrotic in character.

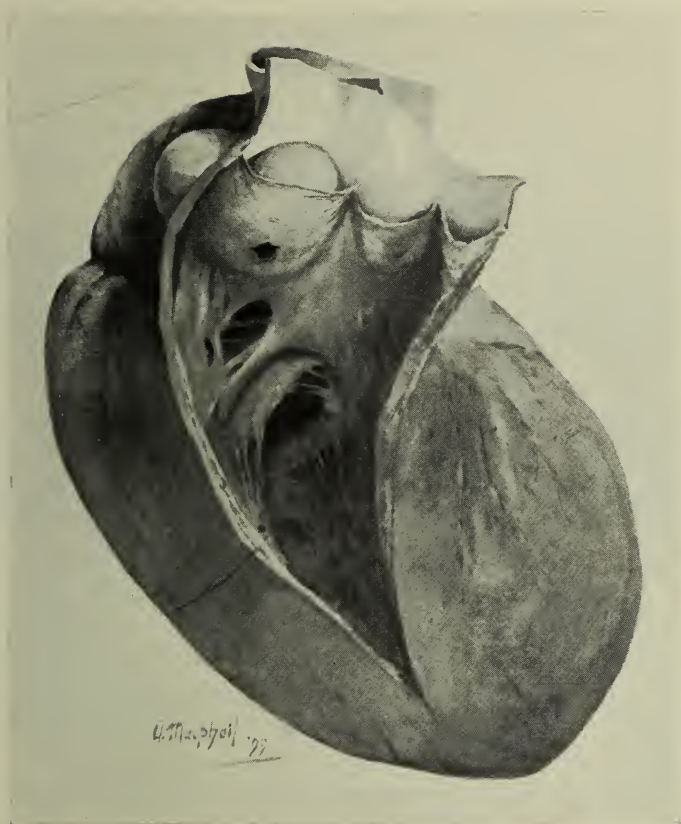
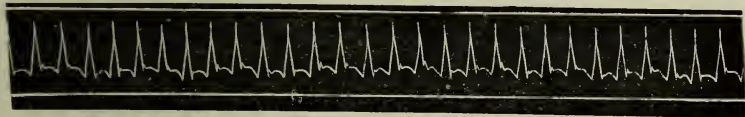
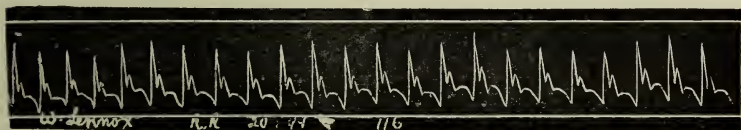


FIG. 2.—The aneurysm as seen from the pulmonary artery, with the somewhat ragged opening into the right ventricle (just below the valve) through which the probe is seen to be passed in Fig. 1. The posterior cusp of the valve of the pulmonary artery is seen stretched over the sac, these valves being in other respects normal. The cut edge of the aorta is seen behind.

In the only case very strictly comparable with this one, contained in the above series (numbered 58 in the list of museum preparations from the Western Infirmary), the brief description given is "Aneurysm of the aorta, projecting into and perforating the pulmonary artery." In this case, which was also under my own care, the aneurysm was much larger than in the present case ("as large as the fist") and, independently of the perforation, must have exercised a very considerable amount of direct pressure, not only upon the



The first of these tracings shows a very characteristic one obtained early after admission, with the very perpendicular up-stroke or primary pulse-wave, the very acute apex, the well-marked tidal wave, and dicrotism as in a case of aortic regurgitation. The second shows the greater degree of approach to hyperdicrotism, at a more advanced period (May 12th), when the rupture may be regarded as fully completed, and the murmurs were in course of developing the much more complicated character described in the last report above cited. The intermediate tracings were not considered characteristic, perhaps owing to the dropsy, which rendered it difficult sometimes to obtain tracings at all, and must have interfered with their definition. The finger, however, for the most part, could still recognize the "water-hammer" character, more or less, throughout.

pulmonary artery, but upon the left auricle and auricular appendage. It was characterized, accordingly, by much more apparently grave symptoms than the present case, and particularly by "great angina pectoris and dyspnoea, with lividity, lasting more than a year; dropsy also, which became extreme towards the close, with considerable (though not sudden) increase of the lividity and the other symptoms, and also pervigilium and orthopnoea. Death was at last sudden, but only after a very lengthened agony." In that case, however, as in the present, it was found exceedingly difficult, if not impossible, in view of the whole facts, to determine at what

precise point in the history the perforation may be supposed to have occurred. As in the present case, the murmurs on admission were V.S. and V.D. in rhythm; and as the aorta presented signs of dilatation in its ascending portion, these were readily enough concluded to have their origin either in the valves, or in an aneurysm of that portion of the vessel. These murmurs, however, became very indistinct during the progress of the case, giving way to a V.S. murmur, which from its localization was regarded as mitral or tricuspid. "Great hypertrophy and dilatation of both ventricles, with venous pulse, became apparent in the course of observation. The liver also became enlarged, the urine scanty, of high specific gravity, and albuminous. The pulse was one of low tension, almost hyper-dicrotous, with an occasional irregularity, but not complete intermission, the low tension increasing as the case proceeded." In my brief remarks appended to the case are these words: "Both the murmurs here were probably aneurysmal, inasmuch as the aortic valves were not diseased. The disappearance of the V.D. in the course of observation is not very easily explained unless as a consequence of the increasingly low arterial tension, which, again, may be referred to the communication with the pulmonary artery. There was no specific fact to indicate the time of the rupture."

In a case which has been frequently referred to, and on which some critical remarks will be found in Walshe, *Diseases of the Heart*, etc., 4th edit., p. 530, Sir Willoughby F. Wade has published (*Med. Chir. Trans.*, vol. xlv., p. 211) what seems to be one of the very few cases of aortic aneurysm in which "a communication with the pulmonary artery was recognized during life by means of physical diagnosis." In this case, as in most of those recorded, a double murmur having more or less resemblance to the usual aortic valvular murmur in rhythm existed, and Dr. Wade lays great stress on the fact that while this double rhythm was evident in the neighbourhood of the conus arteriosus and pulmonary artery, the diastolic element was not propagated to the apex, where, although a blowing murmur attended the first sound, "the cardiac second sound was very distinct and quite natural; no trace of murmur." Upon this,

Dr. Walshe remarks that (according to Dr Wade) "non-conduction of existent diastolic murmur downwards to the heart's apex is the key to the diagnosis of aortic communication with the pulmonary artery. But," he goes on to say, "this inference seems to me very seriously shaken by the fact that in some cases of aortic regurgitation, the attendant diastolic murmur is not transmitted to the left apex, where, on the contrary, a pure second sound is heard; this holds true even of excessively loud basic reflux murmur. Yet more, in a case of aorto-pulmonary communication by W. Roberts, the attendant murmur (a double one) was distinctly heard at the left apex." There is no doubt, I think, that Dr. Walshe is technically right in this criticism, and that Sir Willoughby Wade has laid a stress on this particular diagnostic point which it can by no means bear. At the same time, I am not disposed to consider (as Dr. Walshe apparently does) the whole principle of the diagnosis in Dr. Wade's case, as vitiated by this error. What he actually observed was a double murmur accompanying, or rather replacing, the two cardiac sounds; "that with the second sound being of a hissing character, and so prolonged as to continue till the commencement of the next ventricular systole"; the special seat of these murmurs being "over the cartilage of the fourth left rib." The murmurs were widely distributed, but at this, the spot of their evidently maximum intensity, "a very considerable purring tremor accompanied the second murmur." The propagation of the murmurs along the carotids made it probable that they originated in the aorta, while their maximum intensity at the cartilage of the fourth rib (left) gave the impression that the right side of the heart (conus arteriosus or pulmonary artery) had something also to do with modifying the conduction. The peculiar quality and prolongation of the second murmur, together with its no less unusual distribution, appear to have carried the convictions of the observer by an instinct more sure than his verbal and categorical arguments, towards the conclusion that some very unusual form of lesion, probably aneurysmal, had established a communication between the systemic and pulmonic circulations; and if so, most probably

(on the ground, as stated, of numerical frequency) between the first part of the aorta and the pulmonary artery. It will be found, I think, that the tentative diagnosis indicated in the case which I have here recorded, under circumstances of greater difficulty and obscurity, was in accordance with what has just been stated as bearing on Sir Willoughby Wade's remarkable case. In this latter case, moreover, there was, in the opinion of Dr. Wade, a distinct probability arising from the history, that the first rupture of the aneurysm into the pulmonary artery (there was a second, apparently of more recent date, and attended by much more severe symptoms, into the right ventricle) had originated in a severe effort made to avoid being run over (the patient was a railway porter), which had produced considerable faintness at the time. In my present case, as will have been seen above, there was nothing in the history which, even after knowledge of all the facts, could be construed as having such significance. It is rather remarkable that in only one of the cases in the Glasgow museums is there any history of sudden accident or illness, such as might be expected, and has often been described, as the consequence of rupture of an aneurysm into some other portion of the organs of circulation. This was my case of rupture into the vena cava superior, already alluded to.

In his carefully studied, but no doubt somewhat imaginative, description of a typical case of aneurysmal communication with the pulmonary artery (816, p. 529), Dr. Walshe writes as follows: "If an individual known, or not known, to have been the subject of aortic aneurysm suddenly experience after effort a sensation of something giving way in the cardiac region, feel faint, become pale and exhibit the general characteristics of nervous shock as in the heart, followed by peculiar fluttering in the chest,—if he subsequently suffer from dyspnoea or orthopnoea, more or less cyanotic blueness of the lips, pallor of the face, chilliness, prostration of strength, anxiety, terrible dreams, occasional nausea and vomiting, syncopal and pseudo-epileptic attacks, and become anasarcaous in the lower extremities, while the lungs and liver undergo mechanical engorgement, as proved by percussion,—if all this

coexist with powerful systolic thrill, limited to the second and third left intercostal spaces close to the sternum (lower than this, it is explained in a note, if the heart itself is lowered by hypertrophy), and with loud whirring murmur essentially systolic and intermittent, though sometimes inclining to continuousness (or, it may be, double murmur similarly seated), the diagnosis of sudden communication between the aorta and either the pulmonary artery or right ventricle is warranted."

No one who knows well Dr. Walshe's constant habit of verification, in even the most minute details, of all his own apparently casual statements, can do otherwise than accept this *syndromé*, or "symptomen-complex," as a good working basis for future research, in so far at least as that a case presenting the majority of these phenomena, or the more important of them, in the succession here referred to might fairly be expected to show on a post-mortem examination the lesion, or lesions, here assigned to the description. But the case which I have here placed on record shows indisputably—*first*, that there may be no sudden shock or sense of "giving way" with or without effort; *secondly*, there may be no orthopnoea at all (this was most carefully attested in my case up to nearly the end) and certainly not more, probably much less, of dyspnoea than attends the great majority of ordinary cardiac diseases; *thirdly*, cyanosis may be quite inconsiderable, and also a phenomenon of very late occurrence, almost a part of the agony; *fourthly*, "syncopal and pseudo-epileptic attacks" may be altogether absent throughout. In fact, it may be quite impossible *from the symptoms alone*, even after the facts have been suspected during life and verified after death, to say at what period in the case the rupture has taken place.

As regards the physical signs, my own experience and reading incline me to affirm that the murmurs in such cases will usually be double, the V.D. element not unfrequently predominating, as in Sir Willoughby Wade's case, and very harsh, whether or not accompanied by thrill (Dr. Walshe probably dwells too much on the "systolic" element in these

cases). In the interesting and well-recorded case of the late Sir Wm. Roberts (*British Medical Journal*, 1868, vol. i., p. 421), quoted by Walshe (but not as regards this particular point), the description is as follows: "A loud harsh double murmur was heard at the mid-sternal base; or, rather, it was a single murmur consisting of two parts and covering completely both sounds. This murmur diminished rapidly in intensity towards the apex, where, however, both parts of it were distinctly audible. It was not heard beyond the apex. It was heard in moderate intensity at the ensiform cartilage. It was well conducted up the aorta and into the great vessels at the root of the neck. But the murmur was heard far more loudly at the upper margin of the cardiac dulness, midway between the middle line and left nipple. At this spot it was excessively loud, harsh, and rasping; it gave the impression of being produced very near the ear. It could even be heard by the naked ear at a distance of an inch from the surface of the chest. It diminished in intensity in all directions from this centre. Over the body of the ventricle it *seemed so superficial that it deceitfully resembled a pericardial friction sound.*" I have emphasized this last statement in order that it may be compared with the notes, given above in full from my hospital journal, as to the later stages of the murmurs in the case here recorded. The question of exocardial origin, which ought always to be present to the mind in cases of very complex or anomalous murmurs, is there also raised, but only to be again dismissed, as in Sir Wm. Roberts' case.

On the whole, this description, as well as that of Sir Willoughby Wade and my own personal experience, tends to the conclusion, that while the murmurs in these cases often present a general resemblance to those of aortic valve disease, there are usually details of differentiation which, if carefully studied, will lead up to a different diagnosis, and it may very probably be the case as (following Dr. Sansom) I have assumed in the article on aneurysm of the aorta in Clifford Allbutt's *System*, vol. vi. (p. 399), that, when the opening is into the right auricle or superior cava, the murmurs will tend

towards the right of the sternum, while, when the opening is into the pulmonary artery or conus arteriosus, they will be more definitely heard towards the left. The approach to continuity of the murmurs, also discussed in that article, is well illustrated in the present case, as well as in that of Sir Wm. Roberts, although it is perhaps only in cases of communication with the great veins that we may expect, if at all, the state of matters described by Dr. Ord: "a long continuous humming murmur, never ceasing, but varying in intensity, more sonorous during systole, fainter during diastole, conducted into the neck, and heard over the whole right side of the chest posteriorly." (See article on Aneurysm above alluded to, p 399.)

It remains to be stated, as a negative point of some importance, that murmurs of the above characteristic types may be absent, or unrecognizable, in some cases of aneurysms, either opening into the vena cava, or into the right side of the heart and pulmonary artery. In the other case of rupture into the pulmonary artery, reported in abstract in the *Transactions of the Pathological and Clinical Society*, murmurs which in the main suggested aortic valvular disease, with regurgitation, were present at the earlier observations, and afterwards completely or almost completely disappeared, leaving only a murmur, V.S. in rhythm, near the apex, which might have been, and was regarded as being probably of mitral or tricuspid origin; and as the symptoms were those in general of advanced cardiac disease, with dilatation and hypertrophy of both ventricles, the disappearance of both, but especially of the V.D. murmur, can only be accounted for by the greatly diminished arterial tension, which was further evidenced by the markedly hyperdiastolic character of the pulse at the wrist. The murmurs, too, were regarded in this case (after the P.M. examination) as of directly aneurysmal origin, inasmuch as the aortic valves at least were found to be intact, although it is possible, of course, that the protrusion of the aneurysm into the pulmonary artery, and the strain to which the valves of that vessel were subjected, may have had something to do with their having origin in that vessel.

Cyanosis, too, was much more evident in that case than in the one to which this article is chiefly devoted.

I wish, before closing the present paper, to allude very briefly to yet another case occurring a few months later than the present, and recorded, with illustrations, in the *Glasgow Medical Journal*, vol. xlix., 1898, p. 195 (Glasgow Pathological and Clinical Society, 13th December, 1897). In this case a small aneurysm, with endocarditis, had arisen in connection with the cusps of the aortic valve (which was of course incompetent), and had ultimately caused obstruction in the pulmonic circulation by pouting into the infundibulum of the right ventricle. There was in this case no rupture into the right cavities, but (singular to state) the history was much more conformable to the idea of such rupture, and to Dr. Walshe's typical *syndromé*, than in the case on which this article is founded. There was a distinct history, not only of strain eight weeks before admission, but of a sudden development of cardiac symptoms, especially "an uncomfortable beating of the heart" *immediately* after the strain. Anginous attacks, aggravated very greatly by the slightest exertion and by alcohol, succeeded, and were maintained after this throughout the observation of the case. Tachycardia was so marked a feature of the illness (also throughout) as to excite particular attention and to determine treatment, which, however, though of the most varied kind, was almost entirely ineffectual. There was only slight oedema, and the urine was very slightly albuminous. Temperature was normal. Death was sudden, about six weeks after the apparent origin of the illness (the strain as above). There was no history of rheumatism, and none of syphilis; but it is worthy of notice that the patient's habits were far from temperate; and as he went up for a month's training with the militia after the supposed beginning of the illness, he had abundant opportunities of observing for himself the effects not only of exertion, but of even moderate amounts of alcohol, in increasing his distress. The murmurs in this case were extremely difficult to define, but the one chiefly in evidence was regarded as probably V.D. in rhythm, and was heard best to the left of the sternum rather below

the pulmonic cartilage. From the whole of the facts the following diagnosis was formed during life, and expressed to the clinical class, founding mainly on the persistent and well-marked tachycardia, and the almost indescribable difficulties in detail with regard to the interpretation of the murmurs: "The physical signs were those mainly of the valvular disease. There was, however, a well-defined suspicion of aneurysm, embodied in the clinical abstract used for lecture purposes, and stated in advance at the post-mortem examination. This suspicion arose from certain clinical peculiarities of detail: (a) Anomalies of murmur and sound; (b) suspected dulness at base (doubtful, however, and contradicted in a second report); (c) marked tachycardia, in excess of what is to be expected in a lesion of the aortic valves. The incompetency of the valve explained (P.M.) the double murmur, but there was this *something* in the case which vaguely suggested more.

It appears, therefore, that while the main facts of Dr. Walshe's symptomatic *syndromé* as to the history may be wanting in cases of actual rupture, the most striking of these facts may appear, in a way not unlikely to mislead, in cases where no actual rupture has taken place.

[I desire to take this opportunity of noticing (although not strictly in accordance with the title of this communication) a most valuable paper in the *International Journal of the Medical Sciences* for October, 1890, by the late Dr. Pepper of Philadelphia, U.S., and Dr. J. P. Crozier Griffith, on "Varicose Aneurisms of the Aorta and Superior Vena Cava." This paper altogether escaped my notice when engaged on the article on Aneurysm for Dr. Clifford Allbutt; but from the detailed personal record of one case, and the careful reference to and abstract of twenty-eight other cases presumed to be all that were available in medical literature up to the date in question, this painstaking investigation assumes very high rank in the literature of the subject. I infer from a reading of this memoir, as indeed is stated above: (1) That the diagnosis of rupture into the vena cava superior is usually easy as compared with that into the pulmonary artery; (2) that the sudden onset, indicating the exact time of the rupture, is a much more

frequent and prominent fact in the former than in the latter class of cases (the most distinctive symptom being the dropsy localized in the upper half of the body); (3) that cyanosis in a high degree, in the upper part of the body at least, and accompanying the dropsy, is much more characteristic of rupture into the V.C. than into the pulmonary artery, or even into the right cavities of the heart.]

FOUR CASES ILLUSTRATIVE OF DIFFICULTIES IN THE DIAGNOSIS OF ANEURYSM OF THE AORTA.

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THE four cases in this paper have been selected for publication to illustrate various facts in connection with aortic aneurysm. They may be divided into two groups. In the first (Cases 1 and 2) the descending aorta was the seat of the tumour, which had greatly eroded the bodies of several vertebrae, and ultimately ruptured, but in the one case pain was the predominant feature, in the other it was only a late symptom. In the second group (Cases 3 and 4) the difficulty of diagnosis between aneurysm and mediastinal tumour of other origin is well illustrated, in cases in which shiverings, high fever, and expectoration of pus were dominant features in the diagnosis.

CASE 1.

Aneurysm at junction of thoracic and abdominal aorta giving rise to diagnosis of caries of vertebrae; rupture of aneurysm below the diaphragm.

John C., the subject of this aneurysm, a ship's carpenter, 29 years of age, suffered so much from pain in the small of the back that he was sent into the surgical side of the Infirmary as a case of spinal disease.

In May, 1895, he was suddenly seized, when in apparent good health, with pain in the lumbar region of the spine. On the previous day he had been employed in lifting very heavy

weights, a thing quite unusual with him. The pain was so severe that he was confined to his bunk for 30 days, and not able to resume work for other 14 days. For about four weeks he remained quite free from pain, but had thereafter an attack of three weeks' duration. There was then an interval of two or three weeks during which the pain was absent; but, since that time, there had been very little intermission. About the middle of April, 1896, he was working in a stooping position, which aggravated the pain and made it more constant. At the end of April he had to give up work, and he entered the Infirmary in May, coming under my care at the end of June.

About the beginning of May he had for the first time observed a distinct pulsation in the abdomen, but he had felt no pain there except on pressure.

He had lost flesh to a considerable extent (from $11\frac{1}{2}$ st. to 9 st. 3 lb.), and was gradually growing weaker, slight exertion causing fatigue and breathlessness. He had had no cough or spit, no palpitation, and no oedema anywhere.

The pain was situated in the lumbar region, on either side of the spine, but more severe on the left side, and was of a dull, aching character. At times it passed downwards and forwards into the iliac regions, but not down the legs. There was marked tenderness on each side of the spine at the seat of pain, especially on the left side, and considerable tenderness on percussion over the lumbar vertebrae, chiefly over the second. Pressure upward on the gluteal region also caused much pain. His back felt very weak when he stood or sat up without support.

On examination of the abdomen marked pulsation was detected in the aorta at and above the umbilicus and a pressure murmur with the stethoscope, but there was no excentric pulsation and no localized dilatation of the vessel. There was a doubtful feeling of resistance and dulness in the left lumbar region, which was regarded as probably due to faecal accumulation.

There was no loss of motor power or of sensation in the legs; the knee-jerks were very pronounced, and there was

a degree of ankle clonus on both sides. There was no trouble with the bowels or bladder.

There was no distinct cardiac apex beat; the dulness was small; the sounds were free from murmur. The pulse numbered 50, and was of good quality; the radial pulses were equal. The temperature was not febrile. No abnormality was detected on examination of the lungs, the liver, or the kidneys.

There was a history of several attacks of ague when abroad; of a hard chancre five years prior to admission; and of a left empyema operated upon by resection of rib (with discharge of some three pints of bloody fluid followed in a fortnight by pus) two and a half years before admission. There was no tubercular family history.

The diagnosis was believed to lie between some affection of the vertebrae (caries) and an aneurysm. He was treated by the combined iodides and bromides, and kept under observation for about a month, when he was again transferred to the surgical side. In the end of July he had a rigor, with a rise of his temperature (hitherto generally subnormal) to 101° , the febrile condition lasting for two days. Another rigor, with a temperature of 102° , occurred early in August, but the febrile condition lasted only for one day. He remained in hospital till near the end of August, his pain being relieved by injections of morphine. As nothing had arisen to confirm the diagnosis of aneurysm, a spinal jacket was applied in September, with apparent improvement for a few days, so that he was able to walk about. There was soon, however, a return of the pain with greater severity than ever, and more in front than formerly.

He was readmitted under my care on 3rd November, 1896, greatly emaciated and suffering much more severely than during his previous residence. The abdomen was flat, but not retracted; the whole of its left side was dull, with a feeling of increased resistance which extended some distance down the anterior aspect of the thigh (Fig. 1). There was little or no undue pulsation of the abdominal aorta, but a systolic murmur was audible over it. When lying on his

face it was evident that there was a great bulging of the left side, extending from the 9th dorsal vertebra to the sacrum, the centre of greatest prominence being at the level of the 11th dorsal vertebra, $2\frac{1}{2}$ inches from the middle line. Dulness commenced at the level of the 7th dorsal spine, extended downwards to the crest of the ilium, and was

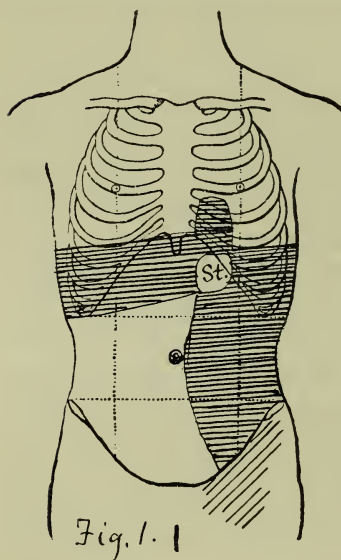


FIG. 1.—Percussion dulness, 5th November.
St. indicates stomach note.

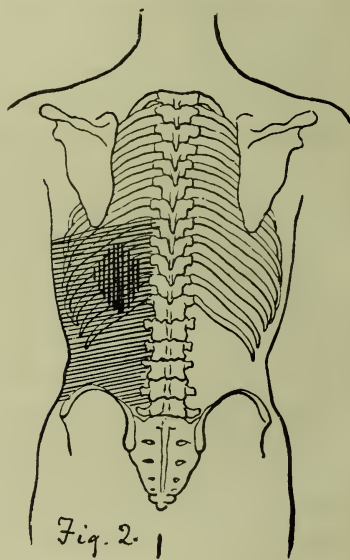


FIG. 2.—Percussion dulness, 5th Nov.
The deeper shading indicates greatest bulging.

continuous with that described in front. Over the greatest bulging, *i.e.* over 10th, 11th, and 12th ribs behind, there was marked pulsation, but not excentric (Fig. 2). A systolic murmur was audible on both sides of the spine over a wide area. There was no apparent difference in the femoral arteries.

There was no tenderness on percussion of the vertebrae and no sign of any interference with the spinal cord. There were no tumours on any of the bones.

The decubitus was on the left side tending towards the prone position, with the right thigh flexed and the left extended.

The further course of the case may be briefly told, as it progressed to a fatal issue.

The pain was so severe that he often required as much as 10 grains of morphine (hypodermically) in the 24 hours, and even then he had little sleep. Emaciation was progressive, and ultimately very extreme. He was pale when readmitted, but the pallor became suddenly more marked on 7th November, and again much more marked during the

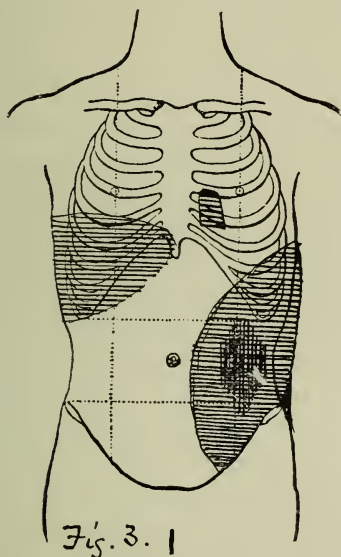


FIG. 3.—Percussion dulness, 21st November. The deeper shading indicates greatest bulging.

last 24 hours of his life. During the whole of this period of residence his temperature oscillated from about normal to 100° or more, the actual maximum being 102.4° ; there was no day without one or more febrile temperatures.

The swelling in the left side increased, and was associated with a degree of yellowish discolouration of the skin and with oedema. The swelling was elastic, but scarcely fluctuant, and even in the abdomen it presented some pulsation (Fig. 3). Oedema of the left foot occurred, without any pain in the leg or thigh, and without any diminution of the pulse in the

posterior tibial artery. Later there was slight oedema of the right foot also.

The distress and exhaustion in this case were extreme. He died on 25th November, 1896.

The heart, which weighed nine ounces, presented healthy appearances, and all its valves were normal.

The right lung was somewhat adherent to the chest wall by old fibrous adhesions. The left lung was very adherent over the outer part of the lower lobe, where the pleura was considerably thickened opposite the scar of the old operation wound.

On removing the stomach and intestines, the parietal peritoneum was seen to be pushed forwards by a large haemorrhage, extending from the diaphragm to Poupart's ligament on the left side, and also on either side of the middle line behind the kidneys, the aorta, and the vena cava. This haemorrhage had arisen from an aneurysm springing from the posterior wall of the aorta just above and behind the pillars of the diaphragm. The aneurysm had first been of comparatively small size, lined with a fairly smooth, but somewhat atheromatous membrane. This sac had ruptured and given rise to a secondary sac of large size which lay behind the left kidney, its walls being formed of fairly firm, stratified clot. This false sac had again given way with the production of extensive haemorrhage behind the parietal peritoneum.

The bodies of the last two dorsal and of the first lumbar vertebrae were deeply eroded, and the last two ribs and the left transverse process of the first lumbar vertebra were also eroded.

Nothing else was observed at the examination calling for note.

This case is of interest from various points of view.

The diagnosis was for a long time doubtful. That there was some pressure on the vertebrae could not be doubted, but whether this was due to an aneurysm or to caries with abscess was uncertain. The occurrence of rigors with pyrexia, the pain and difficulty experienced in sitting up or in trying to stand, and the increase of the knee-jerks with the

presence of ankle clonus tended to the diagnosis of caries of the vertebrae, but the absence of motor and sensory paralyses, etc., seemed to show that there was no pressure on the cord, and the excessive severity of the pain was in itself an argument against caries of the vertebrae. The diagnosis ultimately had to rest upon our knowledge that aneurysm of the aorta is frequently the cause of such pain as was complained of here, the absence of evidences of any other effective cause, and, ultimately, the occurrence of internal haemorrhage. The pulsation that was present when he came under observation was of the kind met with in many affections, being a simple pulsation and not excentric; and even this pulsation disappeared as the case advanced, no doubt masked by the great haemorrhage. Even the pulsation in the tumour that formed in the back was not excentric, but it was suggestive of the presence of blood in the pleural cavity and therefore a valuable aid. The systolic murmur present was too widely spread to be of much assistance, but the fact that it increased in intensity as the case progressed might have been regarded as significant.

Attention should be directed to the prominence of pain as a symptom in this case. For a long time it constituted the only complaint, and after the first few months, when it was occasionally absent for some weeks at a time, it became persistent, but liable to paroxysms. Its severity was extreme, and was no doubt in large part due to erosion of the vertebrae, but in a considerable measure also to the site of origin of the aneurysm, as it is known that aneurysms about the crura of the diaphragm are intensely painful.

The total duration of the case would, at first sight, seem easily determined, as the statement of the patient, a very intelligent man, definitely assigned the origin of the disease to a strain in May, 1895. Such a duration, 18 months, is unusually short, especially when we take into account the long periods of rest in bed with iodide of potassium, by which he was treated. The questions may, therefore, be raised whether the strain in 1895 did not simply aggravate an aneurysm already existing; and, if so, whether the bloody

fluid, undoubtedly removed from the left pleural cavity about the end of 1892, had not been derived from leakage of such aneurysm. If the latter view could be sustained, this case would prove a most interesting instance of recovery from aneurysmal haemorrhage with maintenance of good health for an exceptional length of time.

Even if this explanation of that haemothorax cannot be received, the clinical and the pathological facts indicate that life was maintained for a long time after haemorrhage had commenced. On admission there was a sense of resistance in the left lumbar region, not considered of much importance at the moment, but not unlikely to have been due to the presence of blood behind the left kidney. Haemorrhage had probably already taken place, and had found its way downwards to that situation in which an artificial sac was formed, and, where at a later date rupture again occurred, as evidenced clinically by attacks of pallor and by increasing bulging and resistance. The post-mortem facts clearly indicated repeated haemorrhage, and the highly stratified clots showed an old and marked attempt at arrest of the haemorrhage and cure of the disease.

CASE 2.

Aneurysm of the descending aorta : oozing into the left pleura and the peritoneal cavity.

This case, though very imperfectly recorded, is of interest in connection with the preceding one.

The patient, 42 years of age, was admitted to the Infirmary on 7th November, 1893, and died on the 11th. He complained of pain in the abdomen, and was obviously very ill.

According to his own statement he had always been healthy and strong up till a fortnight before admission. On the 24th of October, while at his work as a hammerman, he was seized with a severe pain in the gastric region which gradually spread over the rest of the abdomen. Thinking it was cramp, he took some brandy, but without obtaining any relief. The

pain was so severe that it took him two hours to walk home, a distance of about a mile. He went to bed and remained there for a week, during which the pain was never absent and sometimes paroxysmally severe. He had a little vomiting, but no looseness of the bowels. He had no medical attendance, but treated himself with rest, light diet, mustard poultices to the abdomen, and seidlitz powders. At the end of a week the pain was gone, but he did not resume work till the morning of 7th November. When he had been at work for a couple of hours the pain returned, and became so severe that he was brought to the Infirmary.

The pain was situated in the right hypochondriac region, and was more severe than it had ever been before. His bowels were somewhat loose, the motions being watery and faecal. There was vomiting, but only after taking fluids (no solids given). He had a rather severe cough, but no spit; the cough seemed to be induced by swallowing. He was pale, but not emaciated.

On admission the abdomen was tense, the muscles being rigid, and there was considerable tenderness below the ensiform cartilage. There was no evidence of fluid in the peritoneum, no dulness, and no enlargement of liver or spleen.

Nothing abnormal was detected on percussion over the lungs, but the respiratory murmur was accompanied by many loud sonorous râles all over the chest. V.R. and V.F. were normal.

There was great pulsation over the praecordial area, the point of greatest impulse being to the left of the nipple line in the fifth intercostal space. The cardiac dulness was somewhat enlarged to the left, and a loud rough systolic murmur was heard, loudest at the apex, but audible over a wide area. The cardiac action was irregular and intermittent; many of the cardiac impulses were not conveyed to the radial pulse, which was soft and rapid.

There was an area of dulness, about one inch in breadth, extending from the cardiac dulness to the suprasternal notch. There was, however, no pulsation over it.

There was no difference in the pupils or in the pulses, but his cough was somewhat laryngeal, and he had some dysphagia.

On 9th November a degree of dulness and increased resistance were made out on the right side of the abdomen, extending downwards from the hepatic dulness.

During the time he was under observation he had numerous attacks of dyspnoea, and he became quite unable to swallow anything. His temperature, 97° on admission, rose to 100° on the day of his death. He was never in a condition in which he could be subjected to much examination.

In this case the diagnosis naturally could not be a definite one, but it was clear that there was something abnormal both in the thorax and in the abdomen.

At the post-mortem examination, "on opening the abdomen, a small but quite distinct trace of blood was found covering the loops of intestine, particularly those lying in the pelvis. The lower part of the omentum was covered with a thin layer of blood. On opening the chest, the left pleural cavity was found to contain 14 or 15 ounces of thin blood. On turning forward the left lung, a bulging, fluctuant, elongated swelling was found in the posterior mediastinum, extending from the level of the sterno-clavicular articulation to the diaphragm, and occupying the region of the descending aorta." This swelling was found to be a large aneurysm, which "had evidently become diffused into the tissues of the posterior mediastinum without actually rupturing the pleura or the pericardium. The blood had thus made its way through the posterior openings of the diaphragm into the region of the abdomen, infiltrating the tissues behind the peritoneum, and also extending for some distance on to the anterior wall of the stomach. This infiltration of blood, which lay immediately behind the cardiac end of the stomach, was so abundant as to at first suggest the possibility of a second aneurysm of the abdominal aorta, but that part of the vessel, beyond very slight atheroma, presented no abnormality throughout its whole extent. It was clear that the traces of haemorrhage met with in other parts of the peritoneum had originated here. The aneurysmal dilatation ceased abruptly at the

diaphragmatic opening. The sixth, seventh, and eighth dorsal vertebrae were much eroded by the pressure of the aneurysm, and this was probably the original site of the tumour before it began to diffuse itself."

The heart was not enlarged. The lungs were somewhat emphysematous, but otherwise presented no abnormality beyond some oedema and hypostatic engorgement, while the bronchi of the left lung contained a pretty abundant purulent secretion. The other organs were normal.

With the results of this examination before us, it is of importance to note the facts as to pain, so far as we could get them. Notwithstanding that erosion of the vertebrae must have been going on for a long time, there was no history of pain in the back at any time. Even when pain did set in, it was not referred to the region of the tumour, but to the abdomen, and it was almost certainly caused by the bleeding into the peritoneal cavity. I am quite prepared to admit that the history was defective, and that this patient probably had had attacks of pain about which he had forgotten; but his well-nourished condition and his being able for heavy work up till a recent period satisfied me that he could not have suffered from severe and continuous pain. This case would seem, therefore, to prove that erosion of the vertebrae may go on without much pain, and that probably the situation of the erosion, involving as it does the nature of the structures on which the aneurysm impinges, must have a good deal to do with the amount and nature of the pain.

This case also illustrates the tendency of aneurysm to rupture and to have the haemorrhage confined, with later rupture and fresh haemorrhage. Along what should have been posterior wall of the aorta there was an old stratified clot, with recent haemorrhage outside of it and in the pleural cavity, etc. A serious rupture had no doubt occurred on the day of his admission to the Infirmary.

The absence of evidence of rupture of the pleura would seem to indicate that oozing into that cavity to a considerable extent may take place from rupture of an aneurysm in the

posterior mediastinum, a fact of some importance in connection with what has already been recorded in the previous case in regard to a pleural haemorrhage.

The date of the first rupture cannot be stated with certainty, but it probably was not very long before his admission. Not improbably it was on the day when he first felt pain, and in that case the duration of life thereafter was short. The duration of the aneurysm itself could only be guessed at vaguely.

CASE 3.

Aneurysm of the arch of the aorta, with attacks of severe dyspnoea, high temperature, and expectoration of pus, causing a mistaken diagnosis of mediastinal abscess.

Early in September, 1898, there was admitted into the Royal Infirmary under my care a man, 38 years of age, a tile-layer to trade, complaining of great shortness of breath of three months' duration. For some time before the breathlessness attracted his attention he had had a slight pain about the right shoulder.

His illness was of very gradual onset, the shortness of breath troubling him at first only on exertion, and slowly increasing in severity as time wore on. By the time of his admission this symptom had become severe, causing him great distress, much aggravated by even slight exertion. Pain had not been a prominent symptom, was of a dull aching character, increased by coughing, and located about the region of the right nipple and of the right scapula; it never shot down the arm.

He had been troubled a good deal by cough, often absent for days, at other times very annoying and accompanied by a peculiar sound which made people in the street turn round and look at him when they heard it. Very little spit accompanied his cough, but he had frequently seen streaks of blood in it. For about five weeks he had had some difficulty in swallowing solid food.

He had been a heavy drinker, but had never had syphilis. His work was not heavy.

He was a strongly-built, well-nourished man. His colour was dusky, the lips and nails being livid. The respiration was normal in rate, but there was orthopnoea. Inspiration was stridulous, and the cough brassy, but no aphonia. The pupils were somewhat unequal, the left being slightly contracted, but both reacted well to light and in accommodation.

There seemed to be slight bulging of the left side below the clavicle, with undue prominence of the second costal cartilage. There was no enlargement of the superficial veins of the thorax or arms, but the veins of the neck were rather full. There was no pulsation in the supra-sternal notch, and no impulse could be detected on palpation over the manubrium sterni or the upper intercostal spaces, but on testing for tracheal tugging there was a very distinct pulsation made out, if not true tugging. The trachea was slightly deflected to the right.

There was a dull area beneath the manubrium sterni, and extending far on either side of it up to the clavicles, but its exact limits could not be laid down. This dulness did not reach as far down as the cardiac dulness, which was small. There was practically no cardiac apex impulse. The sounds were everywhere free from murmur, and they were not accentuated over the praecordial area, but there was some accentuation of the second sound over the manubrium. Only slight difference was made out by the fingers in the radial pulses, but the sphygmograph showed the right to be smaller and of higher tension than the left. There was no oedema of the face, neck, or arms, and no enlargement of lymphatic glands in the neck. The respiratory murmur could scarcely be heard in front, and behind it was feeble on both sides.

Such were the main facts on admission, so that a provisional diagnosis of aneurysm was made, and he was put upon 30 grains of iodide of potassium per day, with inhalations of oxygen when required for his breathing.

He was under treatment until 24th December, when he left at his own request, greatly improved.

During his residence in hospital he had many extremely severe and protracted attacks of dyspnoea, during which his temperature always rose high (maximum $103\cdot4^{\circ}$), and which were generally associated with an expectoration (often 10 oz. in 24 hours) of pus, the latter taking the place of the mucus of which his spit usually consisted. On the first occasion the pus was extremely offensive, but in later attacks the bad odour was absent. Occasionally the spit was blood-stained.

On the morning of 1st October he was seen in one of his bad attacks which had come on late the previous evening. His distress was extreme, so that he could not sit or lie in one position for more than a minute or so at a time, and he was wandering in his talk. This distress was evidently due to the fact that air was not entering a large portion of the lungs, and it was noted that this difficulty did not seem to arise at the larynx (although the left cord was partially paralysed), but from pressure on the trachea. A hurried examination of his back discovered great weakness of the respiratory murmur from the interscapular space downwards on both sides. The sputum was considerable, and consisted almost entirely of blood-stained mucus, quite free from bad odour. He felt cold, but his temperature was $102\cdot6^{\circ}$. Lividity was very marked. Oxygen, nitrite of amyl, injections of sulphuric ether, etc., were tried without the slightest benefit. As a last resource, and because he looked as if he would die unless relieved, one-sixth of a grain of morphine was given hypodermically, which acted almost at once and extremely well. In all such attacks at a later date, the morphine was employed at once, and always with good effect. Following the above attack, there was an expectoration of several ounces of pure pus.

Occasionally a slight systolic murmur was heard over the manubrium, but it was more commonly absent. The general physical facts varied little, *i.e.* the dulness, the R.M., the pulses and pupils, etc.

Between the severe attacks of dyspnoea he improved greatly, his temperature being normal or subnormal, and his respirations almost down to normal, and he was ultimately able to go

down stairs and to come up again (three storeys) without much difficulty.

This case was shown at a demonstration to the Eastern Medical Society,¹ when it was stated that, while many of the facts pointed to aneurysm, the attacks of shivering, with high temperature, and excessive dyspnoea followed by expectoration of pus, at first foetid, indicated rather "the existence of a mediastinal glandular tumour in which there is a suppurating cavity, the retention of the contents of which causes the urgent dyspnoea and high temperatures." That was regarded as the most probable diagnosis, even in the absence of enlarged glands in the neck, and of oedema in the face, neck, or arm, so often associated with such tumours. When at his worst the question of trephining the sternum was even raised, but it was deemed advisable to wait for more definite indications, especially as the morphine relieved the most urgent symptoms so readily.

He left the Infirmary at his own request on 24th December, feeling fairly well, and he died suddenly on the morning of 26th December. He was alone at the time, so that no account of his last attack could be obtained, but there were no indications of haemorrhage.

Permission having been obtained, Dr. Workman made a partial post-mortem examination, and removed the contents of the anterior mediastinum, remarking that there was certainly a tumour. It was only when the tumour was opened that it was found to be an aneurysm of the aorta, just where the ascending passes into the transverse portion, but its cavity was almost filled with laminated clot. The trachea was impinged upon by the tumour, and its mucous membrane was greatly congested and abraded in one or two spots. There was no purulent centre.

The specially interesting facts in this case are the attacks of shivering, with high fever, associated with severe dyspnoea and expectoration of pus, phenomena which are all (with the exception of dyspnoea) unusual in the case of aneurysm.

¹ See *Glasgow Medical Journal*, April, 1899.

Even in the light of the examination after death it is not quite easy to explain their occurrence. It seems to me most likely that they were due, in great part, to sudden aggravations of the tracheal inflammation. But in part, also, they may have had a nervous origin, and this view is supported by the good effect of the morphine. The rapidity with which the injection acted was such as to suggest that the dyspnoea could not be entirely due to pressure on, and congestion of, the trachea, but must in part be due to nervous spasm.

CASE 4.

Mediastinal tumour with many of the signs of aneurysm: when apparently moribund, recovery after profuse expectoration of pus.

In the case just recorded we had the opportunity of completing the diagnosis by post-mortem examination. In the present case the diagnosis is still uncertain, as the patient has fortunately recovered. In their clinical history the two cases present many similar features.

Donald M'M., aged 45, blacksmith, was admitted on 13th October, 1896, complaining of cough and loss of voice, the former of five weeks' and the latter of eight days' duration.

Early in 1894 he began to suffer from pain in the chest. Originating in the middle line in front, it passed round the left side to both shoulder blades, but more especially to the left. The pain never affected the shoulders, the arms, or the neck. It was worse when he lay in bed, and ultimately was so severe that for months he spent the nights sitting by the fireside, and had to have injections of morphine.

In July, 1895, he was in the Western Infirmary; while there he always lay on his back or on his right side, the pain being unbearable when he lay on the left side. About May, 1896, the pain became less severe, and at the time of his admission it had almost disappeared, any that remained being in the left shoulder blade.

The cough was painless and unaccompanied by expectora-

tion; he had never at any time spit blood. The hoarseness and loss of voice came on gradually, reaching their greatest intensity in a couple of days. He had never had palpitation.

But for an attack of sciatica in his left leg in 1892 he had always been a healthy man. He had never had syphilis or gonorrhoea. For 14 years he had had no hard work, as he had been in charge of the smiths' shop; prior to that he had had heavy work.

The patient was a strongly-built man (over 14 stones), inclined to be fat. He lay in bed quite comfortably in any position. Generally he spoke in a whisper, but occasionally his voice had a natural tone except for a slight roughness. Every now and then he gave a harsh vibrating cough of an imperfect character. There was no swelling of the face, arms, or legs. The pupils were equal, and responded to light and in accommodation. There was no visible impulse in the vessels of the neck, nor could pulsation be felt in the jugular fossa. There was no tracheal tugging. The pulse numbered 72 per minute and was regular; sphygmograms showed that the tension was good, but that the right radial gave a less ample tracing than the left; there was no rigidity of the walls of the radial or temporal arteries. Respirations numbered 16 and were quiet.

The apex beat of the heart, not very pronounced, was in the fifth intercostal space in the left nipple line. The cardiac dulness was not enlarged. There was no cardiac murmur, and, though there was some reduplication of the second sound, neither the aortic nor the pulmonic element seemed accentuated. The lungs were normal, but on deep inspiration a snoring sound was heard from the larynx. The urine (sp. gr. 1030) contained neither albumen nor sugar.

Dr. Fullerton kindly examined the throat, and reported as follows: "Both in pharynx and in larynx a catarrhal condition is present. Both true cords are somewhat red and thickened. The left cord is absolutely immobile; the movements of the right cord are unimpaired. There is no localized swelling or thickening seen."

Such was his condition up till 17th October, when his

breathing gradually became difficult and the cough more troublesome. There was no sudden onset of dyspnoea. On the 18th he was rather worse, with slight variations. The lungs presented no difference on percussion on the two sides, and no dulness; the respiratory murmur was equal on the two sides, and there were no râles. In the evening of the 18th his breathing became much more difficult, and he complained of pain in the upper part of the front of the chest. Inhalations of oxygen gave him some relief. About 10 p.m. a very severe attack of dyspnoea occurred, there being as much difficulty with expiration as with inspiration. The pulse ran up to 140. After two hours the attack gradually became less severe.

On the morning of 19th October he was in extreme distress from difficulty in breathing, and very livid; pulse, 140; temperature (previously normal or subnormal), 102.2°. He had then expectoration for the first time, purely mucous, without any trace of blood. A hurried examination of his chest discovered a systolic murmur in the left interscapular space, where also the respiratory murmur was tubular.

He insisted on going home, but he was so ill that he was warned that he might not reach home alive.

The history of pain, the cough, the voice, and the paralysis of the vocal cord led us to the conclusion that we were dealing with an aneurysm of the arch of the aorta. The aggravation of the symptoms was regarded as due to pressure on the trachea, into which it was presumed the aneurysm would burst.

On 1st December, 1896, he presented himself for examination, looking well. His medical adviser, Dr. J. S. Muir, reported that on his return home "signs of pneumonia set in on the left side, and he was very low. An enormous amount of purulent matter was expectorated" (patient says he has been told that this occurred three days after his return home, but he has a very indistinct recollection of what occurred during the first fortnight), "and he has gradually recovered. He is now better than he has been for two years and completely free from pain." The condition of the larynx was the

same as when he left, and the only new phenomena noted were a band of dulness on the left side from the cardiac dulness to the spine, obliterating the gastric clear crescent, with feebleness of the respiratory murmur and marked pleural friction.

When seen again on 31st December, 1896, he had no complaint except of hoarseness. It was then noted that the whole of the left side was full, with bulging and pulsation in the episternal notch. There was no tenderness on pressure and no bulging of the intercostal spaces. The percussion note on the left side was not quite so resonant as that of the right, but the only definite dulness was in the left lateral region and a band of dulness from the cardiac dulness up to the left sterno-clavicular articulation. There was no marked difference in the respiratory murmur on the two sides. There was no cardiac murmur.

On 11th July, 1899, he reported himself as quite well but for hoarseness and cough. He had grown very stout, but he was quite able for ten to fourteen hours' work daily, during which time he was constantly on his feet. At times he had severe attacks of coughing, which sometimes lasted for a whole day, and were associated with a good deal of mucous expectoration, without pus, and only on very rare occasions presenting a few specks of blood. Stooping aggravated both cough and breathing. The left vocal cord was still immobile; voice very hoarse, cough laryngeal, and inspiration stridulous; never dysphagia.

The left side was still unduly full, and somewhat deficient in movement, and its respiratory murmur was rather feeble, especially in front. There was no dulness anywhere over the lungs. During bad attacks of coughing he still felt pain where the friction had been.

There was no enlargement of glands anywhere; no distension of veins in the neck; no pulsation in the episternal notch; no displacement of the trachea; no oedema anywhere.

The cardiac dulness was small, and there was no definite murmur, though the first sound at the fourth left costal

cartilage was so prolonged as almost to amount to a murmur. There was an indefinite dulness about the midsternum, but it did not reach up to the manubrium.

Here, as in the previous case, there occurred a marked accession of dyspnoea, with rise of temperature, followed by expectoration of pus, in this case profuse; but the further progress of the case has rendered the diagnosis of an aneurysm more than doubtful. That there was a mediastinal tumour of some sort is certain, and that suppuration occurred is equally so; but it is not certain whether this was simply a slowly growing abscess, originating, say, in some of the bronchial glands, or whether the suppuration took place in a tumour of another nature. In any case, the present condition of the patient is very satisfactory, though there is no hope that he will regain the use of the vocal cord. That paralysis and the partial obstruction to the entrance of air into the left bronchus are no doubt due to inflammatory adhesions and thickenings which cannot be removed.

FAMILY TENDENCY TO HYPERTROPHIC CIRRHOSIS OF THE LIVER (HANOT'S DISEASE).

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THE occurrence of great enlargement of the liver, associated with jaundice, has long been known; under the name of hypertrophic cirrhosis it is now generally recognized, especially since the teaching of Hanot. With this liver affection we often have enlargement of the spleen, without leukaemia; abdominal dropsy may occur as a complication, but is rare if the comparison is with the regular atrophic cirrhosis. As in the case of the last-named disease, the injurious influence of alcohol may be traced as an etiological factor; but in very many cases of hypertrophic cirrhosis this may be excluded. Of special interest, on this and other accounts, is the study of this form of disease in children, and many cases have now been published in young subjects.

In 1898 I had under my notice a very typical illustration of this disease in a young lad, then 18 years old, a worker in the coal pits; he had been affected for two years at least, and he was not open to the suspicion of alcoholism. A prolonged study of his case, while resident in the Western Infirmary (May 31st, 1898), failed to reveal any new feature. He had very troublesome itching of the skin, which varied much in severity from time to time; and he had had repeated attacks of epistaxis, some of which we witnessed, but no haemorrhages from the stomach or bowels had occurred. The jaundiced

tint was distinct both in the skin and conjunctivae although not extreme; the urine was dark in colour and showed a slight reaction of bile pigment on testing with iodine, very indistinctly with nitric acid, or not at all. The motions were sometimes clay-coloured, at other times natural, without any corresponding variation in the jaundiced tint; no dropsy; enormous enlargement of the liver, which felt hard and smooth; no distinct enlargement of the spleen could be made out, a very definite tumour in the left hypochondrium being regarded as the left lobe of the liver, as it seemed continuous with that organ; no glandular enlargements anywhere.

His previous history was that of frequent attacks of feebleness and of bad health, with some little abscesses, but nothing either in himself or in the family, which was a moderately large one, appeared to suggest syphilis. He seemed, for his age, somewhat under-developed. Under treatment he improved a little, but his condition was not materially altered when he left; indeed, he seemed very much as before when seen again at the Infirmary a year later (May, 1899). He then weighed 7 st. 1 lb., much the same as the year before.

On his return, however, a new point of great importance emerged. His medical attendant, Dr. Muir of Bellshill, reported that the patient's sister, two years older, was similarly affected, and they were sent in together for comparison. The sister presented a striking family resemblance to her brother. She too was under-developed, and her menses had never appeared, although she was 21 years old. She also had suffered from feeble health at least since she was 14 years: her colour was exactly the same as her brother's; there was the same slight reaction of bile pigment in the urine. She too had been troubled with itching of the skin, although not in such a marked form. She also had repeatedly lost blood from the nose. No dropsy existed, and there had been no haemorrhages from the stomach or bowels. In her the liver was greatly enlarged—not quite so extremely as in her brother's case; but in her the spleen was felt to be greatly enlarged. Indeed, on examining the

abdomen, the splenic tumour was more striking than the enlarged liver. No glandular swellings were detected, and there was no alcoholic history. She weighed 7 st. 11 lbs.

An examination of the blood was made by Dr. Carstairs Douglas during the lad's residence in 1898, and again when he returned with his sister. No increase of the white blood corpuscles existed in either case; indeed, some of the estimates made them less than the normal. The number of the red blood corpuscles was about the normal in both patients, and the haemoglobin was not much below par.

This discovery of a second member in the family being affected led Dr. Muir to examine some of the other members of the same family, and he reported that a third member, a younger brother, was also found to have a large liver and spleen. Dr. Muir supplied the following notes of this lad:—Age, 15 years: affected for 18 months; jaundice slight; itching occasional; bleeding from nose sometimes; liver large, extending 3 inches below the ribs; spleen enlarged, extending $3\frac{1}{2}$ inches below the ribs; no history of alcohol. Another brother, still younger, seems a little yellow, but no enlargement of liver or spleen can be detected in his case.

Three members of a family similarly affected with a condition relatively rare in early life led to a search into the literature of cirrhosis of the liver, especially as regards young subjects or family proclivity.

The little memoir of Hanot does not indicate any experience of family tendency in this disease, although he refers to the subjects being weakly and poorly developed (p. 99). In Osler's *Practice of Medicine*, third edition, 1898, p. 574, the writer states, under the heading of "Hypertrophic Cirrhosis," that two of the cases he had seen were in brothers. In the *International Journal of the Medical Sciences* (Oct., 1887, p. 350), in an article on "Hepatic Cirrhosis in Children," Dr. R. P. Howard gives the case of a girl, 9 years old in 1878, affected with a large liver and spleen, with a history of epistaxis, slight pyrexia, and latterly ascites. Her brother became icteric in 1884; he also had enlargement of the liver and spleen, and he also had slight pyrexia and ascites.

Neither of them had leukaemia. Both died, and in both the livers are described, post-mortem, as large and granular.

In the *British Medical Journal* for April 23rd, 1892, p. 858, Mr. Jollye reports two cases in children, a brother and sister, who died with ascites due to atrophic cirrhosis; although a post-mortem examination was obtained only in one case, he presumed the condition was similar in the other. He also quotes Dr. Ormerod's paper, which falls to be noticed hereafter.

The existence of a family tendency to hypertrophic cirrhosis is obviously important, although its significance is still obscure. Of course, if due to syphilis, the explanation might be clear; or, if due to transmission from a drunken parent (see Hanot, p. 99), the disease might well occur in various members of the family. Both of these causes seem fairly to be excluded in the family here referred to.

The resemblance of hypertrophic cirrhosis to what is called "biliary cirrhosis" seems very close. Further, in hypertrophic cirrhosis, we have a disease characterized by jaundice, one occurring in early life, and specially implicating certain families; it may be well, therefore, to consider some of the features of congenital malformation of the bile ducts where these peculiarities are also found.

The occurrence of biliary cirrhosis, with enlargement of the liver and often with enlarged spleen also, is well known in cases of congenital obliteration of the bile ducts. "It seems probable that biliary cirrhosis always occurs if the child lives long enough" (Dr. John Thomson, *Congenital Obliteration of the Bile Ducts*, Edin., 1892, p. 29). Dr. Thomson also says, of such congenital defects, "there is evidently a very remarkable tendency for the disease to occur in more than one child of the same parents" (p. 13). In his tabulation of cases we find one (No. 22) where one twin died with obliteration of the duct, and the other, although jaundiced, recovered. Other cases (No. 9 and No. 33) in his table show recoveries from icterus neonatorum in families where fatal defects of the ducts had occurred. Further, cases are given in table No. 4, where, with similar symptoms proving fatal and in families

affected with this grave form of icterus, the ducts were found to be still pervious. From all this we may infer a great degree of variation as to the severity or extent and also as to the duration of the affection of the bile ducts, or the period in intra-uterine life when the disorder appears. It is also important to notice that Dr. Thomson shows that this defect is sometimes associated with other manifestations of developmental disorder. In my cases of hypertrophic cirrhosis the brother and sister were evidently under-developed, and in particular the girl, although 21 years old, had never menstruated. The remark already quoted from Hanot (p. 99), as to the disease occurring in badly-developed subjects, may be recalled in this connection.

In Dr. Ormerod's paper (*St. Bartholomew's Hospital Reports*, vol. xxvi., 1896) reference is made to the occurrence of cirrhosis of the liver in a young subject associated with a family tendency to some peculiar nervous disorder; but, in the absence of details, not yet published, it is difficult to estimate the value of this group of cases for our present purpose. In the same paper he quotes the experience of Prof. Homén (*Neurologisches Centralblatt*, 1890, p. 514), where two members of a family had this combination of well-marked cirrhosis of the liver and some nervous lesion (in the lenticular nucleus) when examined after death; a third member of the family seemed to be similarly affected. The illnesses ran a chronic course of six or seven years, and proved fatal at the age of 26 and 19 years respectively.

In the case of family cirrhosis we may suppose that a developmental defect occurs in the liver in certain members of the family; the mere fact of the common bile duct being found pervious at death in cases of hypertrophic cirrhosis (Hanot, p. 132) does not exclude the notion of a possible defect or peculiarity in the ducts determining a biliary cirrhosis. We know that very slight obstructions interfere with the flow of bile, and some defect in the lumen of the ducts within the liver itself might lead to disorder. We have instructive analogies in the congenital defect of the bile ducts, already referred to, viz. cases with similar symptoms occurring at

birth, proving fatal, with ducts still pervious; and, on the other hand, cases of recovery in jaundiced infants whose brothers or sisters died with the developmental defect of the ducts. Still further, there is a case of some importance from this point of view communicated by Dr. Fred. J. Smith to the Pathological Society of London (*Pathological Transactions*, vol. xli., p. 154, 1890), where an infant recovered from an attack of icterus neonatorum after a fortnight, but became the victim of hypertrophic cirrhosis, with greatly enlarged spleen, without dropsy, dying at the age of $4\frac{1}{2}$ years. To be compared with this is the family described by Pearson in Underwood's treatise on *Diseases of Children*, 4th ed., 1799 (see Thomson's Table IV., No. 1), where nine children in the family died under a month old, from jaundice, and the tenth died from jaundice also, when six years old; in the post-mortem examination of the eleventh child in this family, who also died of jaundice on the ninth day, the ducts were found to be pervious and the gall bladder full of bile.

Such ideas arise in connection with my three cases of hypertrophic cirrhosis in one family, when compared with other reports showing a family tendency to this disease. It must be remembered, however, that the cases called by this name constitute still a very mixed group. Indeed, it may be, as contended by some, that these enlarged livers might in time become small and granular. The records of Dr Howard's cases, of a brother and a sister, showed large livers in both, but in both the surface was granular.

If attention is called to the question of family tendency in hypertrophic cirrhosis, especially in early life, some further light may arise when more cases are definitely investigated; for it is worthy of notice that in my first case, although the family history was inquired into, nothing of this kind was elicited, and it was only from Dr. Muir's personal knowledge of the family that this important peculiarity was noticed in the second case, and searched for in the third.

REFERENCES.

THE little book of Hanot, *La cirrhose hypertrophique avec ictère chronique*, Paris, 1892, gives a full account of the whole subject, with historical references, including his own thesis, *Sur une forme de cirrhose hypertrophique*, Paris, 1875. There is an elaborate review of the subject, up till 1877, in *The British and Foreign Medico-Chirurgical Review*, July, 1877, "Hypertrophic Cirrhosis of the Liver." An important tabulation of cases of "Cirrhosis of the Liver in Childhood," or early age, is given by W. A. Edwards in the *Archives of Pediatrics* for July, 1890, vol. vii., p. 502, where he also contributes a case of the hypertrophic form. Dr. Mary Putnam Jacobi has an interesting article in the *Archives of Pediatrics*, May, 1889, vol. vi., p. 273, "Case of Cirrhosis of the Liver with Splenic Tumour" (girl 10 years old); in this paper she quotes various authors, amongst others, Laure et Honorat, "Étude sur la cirrhose infantile," *Rev. mens. d. mal. de l'enfance*, t. v., Paris, 1887. (I quote the reference from the *Index Medicus* as I have not seen the original.) Dr. R. Palmer Howard, "On Hepatic Cirrhosis in Children," in *International Journal of the Medical Sciences*, October, 1887, p. 350 (a sister and brother). F. W. Jollye, "Hepatic Cirrhosis occurring in Two Children of the same Family," *British Medical Journal*, April 23rd, 1892, p. 858. Ormerod, "Cirrhosis of the Liver in a Boy," *St. Bartholomew's Hospital Reports*, vol. xxvi., 1890, p. 57; this paper has several points of interest in connection with the question of family tendency, and in particular he gives a reference to Prof. Homén, *Neurologisches Centralblatt*, Bd. ix., 1890, p. 514, referred to above, where two or probably three in a family were affected with cirrhosis of the liver and some rare nervous disease. The title in *Index Medicus* is "Eine eigenthümliche Familienkrankheit unter der Form einer progressiven Dementia mit besonderem anatomischen Befund." Osler's *Practice of Medicine*, 3rd edition, Edinburgh, 1898, p. 574 (two brothers with hypertrophic cirrhosis). The literature of hypertrophic cirrhosis of the liver is now very considerable, and that of cirrhosis of the liver in childhood is also large, as may be found on consulting the *Index-Catalogue* of the Washington Library, and the *Index Medicus*; many of the cases are tabulated in papers quoted above.

AN ANALYTICAL STUDY OF CERTAIN OF THE CLINICAL PHENOMENA OBSERVED IN 112 CON- SECUTIVE CASES OF CHOREA.

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IN this paper I propose to give the results of an analysis of the chief clinical phenomena noted in connection with 112 consecutive cases of chorea minor (St. Vitus' dance—Sydenham's chorea), of which I have preserved more or less extensive notes in my case-books. It is possible that this analysis may not throw very much fresh light upon the disease ; but the larger the number of carefully recorded cases subjected to statistical investigation, the more accurate is our knowledge of the phenomena of the disease likely to become, and this has been the chief reason which has induced me to prepare this analytical review of my notes. The cases were seen in the dispensary of the Glasgow Royal Infirmary, in the out-patient room of the Royal Hospital for Sick Children, Glasgow, and latterly in my wards at the Royal Infirmary. In the laborious work of examining the records and reducing them to statistical form I have gratefully to acknowledge the valuable help I have received from my colleague at the Sick Children's Hospital, Dr. Alice M'Laren and from my house physician at the Infirmary, Dr. Hugh M'Laren. It is perhaps not without importance in an inquiry of this kind that all the cases dealt with have been personally examined and recorded by myself. The results of

the inquiry are likely to be more uniform, and the defects and omissions of the observer more easily detected and allowed for than if the cases analyzed had been recorded by a multiplicity of observers, each with his own peculiar methods of note-taking and examination. I desire also at this point to make it clear that I do not intend to discuss the phenomena of the so-called choreiform affections—tic, habit-spasms, post-hemiplegic chorea, Huntington's chorea—all of which I believe to be totally different, both in clinical course and pathological nature, from the chorea minor of childhood. To discuss these affections along with Sydenham's chorea is, in my opinion, likely to give rise to confusion of thought and to disappointment in treatment.

In presenting the results of my analytical study, I shall arrange the 112 cases in two groups:

A. 87 Cases observed in the Out-patient Room.

B. 25 " " Wards.

Obviously the information at my command was greater in reference to the cases admitted to the wards than it could be in connection with the out-patients. The phenomena of the disease which have been investigated in this inquiry are the following: (1) sex, (2) age, (3) number of attacks, (4) duration of attacks, (5) cause of attacks, (6) family history, (7) association with rheumatism and other morbid states, (8) condition of the heart, (9) condition of the urine, (10) distribution and severity of the movements, (11) time under observation.

A. 87 OUT-PATIENT CASES.

(1) *Sex.*

Of these cases 23 were males and 64 females, *i.e.* about 26·5 per cent. were males and 75·5 per cent. were females. This proportion of females to males is somewhat greater than that given by Osler, who found that in 554 cases about 70 per cent. were females. He remarks that his proportion of females is lower than that given by many authors.

In 31 per cent. of the cases more than one attack of the disease was recorded, a percentage which shows strikingly enough the great tendency chorea has to recur, and the figures show that three, four, and five attacks are not at all uncommon. This percentage of recurrences is less than that given by Osler, who had 41 per cent. of recurrences in 410 cases analysed with special reference to this point.

(4) *Duration of Attacks.*

It is more difficult to show the results of our inquiry into this point in a tabular form. In the first place, it is to be noted that the table only deals with the duration of the attack for which the patient was under treatment, and, secondly, that in 18 out of the 87 cases no information has been preserved in the records. In 36 of the cases I obtained definite information, and in 33 partial or indefinite information, as shown in the table.

A. No information, - - - - - 18 cases.

B. Definite information :

One month's duration and under,	-	7 cases.	} 36 cases.
Two months' ,, ,,	-	11 ,,	
Three ,, ,, ,,	-	13 ,,	
Six ,, ,, ,,	-	4 ,,	
Twelve,, ,, ,,	-	1 case.	

C. Indefinite or partial information :

One month's duration and under,	-	16 cases.	} 33 cases.
Two months' ,, ,,	-	5 ,,	
Three ,, ,, ,,	-	2 ,,	
Six ,, ,, ,,	-	3 ,,	
Twelve,, ,, ,,	-	1 case.	
Twenty-four months' duration and under,	-	2 cases.	
Congenital, - - - -	-	1 case.	}
Very chronic, - - - -	-	3 cases.	

Total, - - - 87 cases.

More than half of the cases lasted under two months, and 15 out of the 69 under three months. No doubt it would have been interesting could the effect of treatment on the duration of the disease have been estimated and tabulated, but this was impossible. The figures, however, show that there is a spontaneous tendency towards the disappearance of the movements.

The following table shows the duration of the individual attacks in 27 cases in which there was more than one attack of the disease:

Duration of the attacks in 27 cases in which there were more than one.

1st attack.	2nd attack.	3rd attack.	4th attack.	5th attack.	6th attack.	No. of Attacks.
4½ months.	Unknown.	2
5 months.	Unknown.	2
3 years.	Unknown.	2
2 months.	Unknown.	2
Some months. }	3 months.	2
3 months.	Unknown.	2
2 months.	3 months.	2
Unknown.	2¼ months.	2
3½ months.	3 months.	2
2 months.	1½ month.	2
1½ month.	Unknown.	2
Slight.	Unknown.	2
1 month.	6 months.	2
1¾ month.	Unknown.	2
3¼ months.	Unknown.	2
Unknown.	3 weeks.	2
Unknown.	2 months.	1 month.	3
Unknown.	Unknown.	1¼ month.	3
1¼ month.	1¾ month.	3 weeks.	3
3 months.	2 months.	3 months.	3
Unknown.	Unknown.	Unknown.	5 months.	4
Unknown.	Unknown.	Unknown.	18 months.	4
4 months.	{ Some months. }	3 months.	Unknown.	Unknown.	...	5
3 months.	1¾ months.	Unknown.	2½ months.	3 months.	...	5
Unknown.	Unknown.	Unknown.	3 months.	Unknown.	...	5
Unknown.	1¼ month.	Unknown.	Unknown.	1¼ month.	...	5
Unknown.	Unknown.	3½ months.	1 year.	1½ month.	1 month.	6

(5) *The Cause of Attacks.*

The association with acute rheumatism and the doctrine that chorea is but one of the many possible manifestations of

the rheumatic diathesis in childhood will be referred to in another part of this paper. Apart from this, however, I have endeavoured from my notes to gain information as to what might be called the emotional factors in the causation of St. Vitus' dance. In only 30 out of the 87 cases have definite statements as to the causation of the attacks been recorded. In at least 26 of these 30 cases the exciting cause may be classed under the heading "Emotional." The four remaining cases were attributed by the parents to the effects of accidental falls, three of the falls being on the head. The following table indicates the different kinds of emotional disturbance to which the chorea was attributed:

Fright, - - - - -	14 cases.
Over-work, - - - - -	2 „
Mental excitement or distress, - - - - -	3 „
Grief, - - - - -	1 case.
Worry over school examinations, - - - - -	3 cases.
Punishment at school, - - - - -	2 „
Scolding, - - - - -	1 case.
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Total, - - - - -	26 cases.

(6) *Family History.*

In the 87 cases there were 31 in which no definite information as to the family history was recorded, leaving 56 in which the family history had been inquired into. In these 56 cases information as to the presence of rheumatism, chorea, or nervous disease in other members of the family was specially sought for. In 30 cases the family history was negative as regards any of these diseases. In 26 cases positive information was obtained, the details of which may be stated as follows: In 18 cases rheumatism had attacked other members of the family; in 11 chorea had occurred; and in 5 some other form of nervous affection. In three of the cases investigated, rheumatism, chorea, and nervous disease had occurred in other members of the same family and in two rheumatism and chorea.

The result of this part of our inquiry, then, is to demonstrate that in almost 50 per cent. of the cases in which the point was specially investigated, a family tendency to rheumatism, chorea, or some other form of nervous disease could be very distinctly made out.

(7) *Association with Rheumatism and other Morbid States.*

The results of the inquiry under this heading are seen at a glance in the following table:

No definite information,	- - -	6 cases.
No history of antecedent rheumatism,	-	46 „
History of antecedent rheumatism,	-	26 „
History of various antecedent diseases,	-	9 „
Total,		- - 87 cases.

It is thus seen that there was no history of antecedent rheumatism in 55 out of the 81 cases in which the matter was inquired into. I believe, however, that the relationship between rheumatism and chorea is much closer than these figures would seem to indicate. It has frequently happened to me to observe a well-marked attack of articular rheumatism follow, often after a considerable interval, an attack of chorea. It seems to me, therefore, that the teaching of Dr. Cheadle, that chorea is but one of several of the manifestations of the rheumatic state in childhood, is strictly borne out by clinical facts. It is because of this intimate association with the rheumatic diathesis that we are justified in regarding Sydenham's chorea as a disease *sui generis*, as a disease quite different both in its clinical manifestations and its pathogenesis from Huntington's hereditary chorea and the various forms of choreiform affections. The adoption of the name "infectious chorea" (*infectiöse chorea*) to signify chorea minor by Professor Wollenberg of Hamburg may be mentioned in support of the opinion of the essential pathology of St. Vitus' dance which I am now advocating.

Of the other morbid states noted to have been associated

with the chorea, in 9 of the cases the following are the particulars:

Bronchitis, - - - - -	2 cases.
Diseased bone, - - - - -	2 „
Convulsions, - - - - -	1 case.
Adenitis, - - - - -	1 „
Idiocy, - - - - -	1 „
Headaches, - - - - -	1 „
Psoriasis, - - - - -	1 „
<hr/>	
Total, - - - - -	9 cases.

(8) *Condition of the Heart.*

In 50 of the 87 cases the heart was stated not to have been affected; and in 5 no definite information as to the condition of that organ was recorded. In 32 cases there was evidence of an abnormal condition of the heart, and the following table shows the state of the cardiac sounds in these:

Accentuated second sound at base, - - -	5 cases.
Prolonged first sound at apex, - - -	5 „
Systolic murmur at apex, - - -	16 „
Presystolic and systolic murmurs at apex, -	3 „
Systolic and diastolic murmurs at apex, -	2 „
Irregularity of cardiac sounds, - - -	1 case.
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Total, - - - - -	32 cases.

These figures show that in 39 per cent. of the cases examined the cardiac condition was abnormal, and if we exclude those in which the second sound was accentuated we get about 31 per cent. in which it may be admitted there was definite evidence of valvular disease.

(9) *Condition of the Urine.*

An analysis of the urine was made at least once in 45 out of the 87 cases at present under review, and gave the following results:

Albumen was detected in - - - - -	4 cases.
Albumen was not detected in - - - - -	41 „
<hr/>	
Total, - - - - -	45 cases.

A note of the specific gravity was recorded in 40 cases.

The average sp. gr. was	-	-	-	-	-	1018.5.
The maximum was	-	-	-	-	-	1033.
The minimum was	-	-	-	-	-	1004.

The reaction of the urine was noted 33 times :

Reaction acid,	-	-	-	-	-	26 cases.
„ alkaline,	-	-	-	-	-	3 „
„ neutral,	-	-	-	-	-	4 „
Total,						33 cases.

The result of this part of the inquiry is to show that in chorea there is on the whole little tendency to derangement of the renal function. One might expect that in a disease characterized by long-continued and often excessive muscular action the specific gravity of the urine would be increased from an excess of waste products being present, but I have not found this to be the case.

(10) *Distribution and Severity of the Movements.*

It is somewhat difficult to show in a satisfactory manner the results of the examination of the records with regard to this point. As to the distribution of the choreic movements, however, some idea may be obtained by classing them under the headings of General, Right-sided, Left-sided. In two cases there was no information.

General,	-	-	-	-	-	39 cases.
Right-sided (chiefly),	-	-	-	-	-	25 „
Left-sided (chiefly),	-	-	-	-	-	21 „
Total,						85 cases.

The only way in which an indication, and that only approximately, of the severity of the choreic movements can be expressed is by classing the cases under the terms severe, moderately severe, and slight. In 5 cases there was no record of this point.

Movements severe,	-	-	-	-	-	23 cases.
Movements moderately severe,	-	-	-	-	-	35 „
Movements slight,	-	-	-	-	-	24 „
Total,						82 cases.

The table shows that cases of moderate severity predominate, and this is probably the usual rule. Of course, as has been said, the statements under this heading can only be approximate, but on the whole the number of cases classed as severe may be taken as unusually high.

(11) *Time under Observation.*

In the work of tabulating the cases an attempt was made to show the time each case had been under observation, but in the end it was found that only the most general statements could be made under this heading. Indeed, all that can be done is to show how many made more than one visit to the dispensary. 31 cases were seen only once; 56 cases made several visits, very many of them having been under treatment for a month or more.

B. 25 CASES OBSERVED IN THE WARDS.

(1) *Sex.*

Of the cases observed in my wards at the Glasgow Royal Infirmary 5 were males and 20 were females, *i.e.* 1 male to every 4 females, as compared with about 1 to 3 in the cases observed in the out-patient room. Here again, then, the rule that chorea is much more frequently met with in girls than in boys is abundantly confirmed.

(2) *Age.*

Arranging the ward cases, like the out-patient ones, in five-yearly periods, we get the following table:

5 years and under,	-	-	-	-	-	0 cases.
6 to 10 years,	-	-	-	-	-	9 „
11 to 15 years,	-	-	-	-	-	11 „
Over 15 years,	-	-	-	-	-	5 „
Total,						<hr/> 25 cases.

Here, as in the former table, it is seen that by far the greatest number of cases occur between the ages of 5 and 15. In the ward cases the proportion over 15 years of age is slightly higher than in the dispensary cases, which, had the proportion been equal, should have given 17 instead of 12 above this age. In the wards there were no cases seen under the age of 5, as against 6 seen under this age in the series of 86 dispensary cases. Both sets of figures then go to prove that under the age of 5 and over that of 15 for a first attack chorea is a rare disease. Of the 25 cases met with in the wards only one of the 5 patients over the age of 15 was suffering from the first attack of the disease; 3 were in the second, and 1 in the third attack, the first attack in all of the cases having occurred a number of years before. The girl suffering from her first attack was between 16 and 17 years of age, and was pregnant.

(3) *Number of Attacks.*

The annexed table shows the number of attacks in the 25 ward cases :

One attack,	-	-	-	-	-	-	-	11 cases.
Two attacks,	-	-	-	-	-	-	-	9 „
Three „	-	-	-	-	-	-	-	3 „
Four „	-	-	-	-	-	-	-	2 „
								—
Total,	-	-	-	-	-	-	-	25 cases.

In 56 per cent. of the present series, as against 31 per cent. of the former, more than one attack of the disease had occurred. We saw that Osler had 41 per cent. of recurrences in 410 cases analysed with reference to this point. In my ward cases the percentage is distinctly greater, but if we take the average of dispensary and ward cases we get 43 per cent. of recurrences, a very close agreement with Osler's results.

(4) *Duration of Attacks.*

In 24 of the 25 cases definite information on this point has been recorded, and is shown in the table :

One month's duration and under,	-	-	-	1 case.
Two months'	"	"	-	5 cases.
Three "	"	"	-	8 "
Six "	"	"	-	7 "
Twelve "	"	"	-	2 "
Three years'	"	"	-	1 case.

An examination of this table shows that a considerably larger proportion of the ward cases lasted more than two months, as compared with those seen in the dispensary, and this probably means that the cases admitted to hospital were on the whole more severe. Here also, though perhaps not so strikingly as in the dispensary cases, the spontaneous tendency to cure is demonstrated, as considerably more than half of the cases lasted three months and under.

The following table shows the duration of the individual attacks in 14 cases in which there were more than one :

Duration of the attacks in 14 cases in which there were more than one.

1st attack.	2nd attack.	3rd attack.	4th attack.	No. of attacks.
Unknown.	5 months.	2
4 or 5 months.	5 months.	2
3 months.	3 months.	2
Unknown.	3 years.	2
12 weeks.	13 weeks.	2
8 months.	12 months.	2
6 months.	5 months.	2
Some months.	6 weeks.	2
7 weeks.	8 weeks.	2
3 months.	3 months.	3 months.	...	3
10 weeks.	8 weeks.	8 weeks.	...	3
4 or 5 months.	10 weeks.	1 month.	...	3
4 months.	A short time.	A short time.	9 weeks.	4
3 months.	6 weeks.	6 weeks.	7 months.	4

(5) *The Cause of Attacks.*

The following table shows the results of the inquiry into the cause of the attacks of chorea in the 25 ward cases:

No cause could be assigned in	-	-	-	16 cases.
Fright " "	-	-	-	7 "
Punishment at school assigned in	-	-	-	1 case.
Pregnancy assigned in	-	-	-	1 "
Total,	-	-	-	<hr/> 25 cases.

The figures in both of my series of cases go then to show that emotional causes, such as fright, are perhaps not so frequent as one would expect in chorea.

(6) *Family History.*

In 23 out of the 25 hospital cases notes of the family history with special reference to the presence of rheumatism, chorea, or nervous disease in other members of the family have been preserved.

In 11 of the cases the family history was quite negative as regards this point.

The following table shows the result as regards the remaining cases:

Rheumatism in family,	-	-	-	-	11 cases.
Chorea in family,	-	-	-	-	1 case.
Nervous disease in family,	-	-	-	-	0 "
Total,	-	-	-	-	<hr/> 12 cases.

The proportion of cases in which a family tendency to rheumatism or chorea was present is slightly greater in this than in the former series (dispensary cases), but the difference is not great. In the ward cases the mother had suffered from rheumatism in 6, the father in 4, a brother in 1, and a sister in 1. In some of the cases, however, rheumatism had attacked several members of the same family. A mother and some of her children had been attacked in 2 cases; the father and mother in 2 cases; a father and son in 1 case. The mother was the only member of the family attacked in 3 cases, and

the father alone in 2 cases. We see then, as in the previous analysis, that in about 50 per cent. of the cases a distinct tendency to rheumatism in the family can be made out in chorea. This, I think, lends strong support to the opinion of Cheadle that chorea is essentially a rheumatic manifestation.

(7) *Association with Rheumatism and other Morbid States.*

Definite information on this point was obtained in the whole series of cases admitted into the wards, and is tabulated as follows:

No history of antecedent rheumatism,	-	8 cases.
History of antecedent rheumatism,	- -	11 „
History of subcutaneous fibrous nodules,	-	1 case.
History of other morbid states,	- - -	5 cases.
<hr/>		
Total,	- -	25 cases.

In the present series of cases a distinctly larger proportion (almost 50 per cent.) had a definite rheumatic history. The history of fibrous nodules may be regarded as evidence of the presence of rheumatism. My experience with regard to the occurrence of nodules in rheumatism or chorea in childhood is that the phenomenon is decidedly rare in Glasgow, and Osler tells us that the condition is also rare in the United States. I have been on the outlook for it for many years, but the case here included in the table is the only one in which I have met with it.

The following table shows the other morbid states which were associated with the chorea in the present series of cases:

Bronchitis,	- - - - -	1 case.
Albuminuria,	- - - - -	1 „
Scarlet fever,	- - - - -	3 cases.
<hr/>		
Total,	- -	5 cases.

It is well known that rheumatism frequently complicates scarlet fever, and therefore it is not surprising that we should find a history of its association with chorea in a certain number of cases.

(8) *Condition of the Heart.*

In my 25 hospital cases the heart was found to be definitely affected in 15, and not affected in 10 cases, again a distinctly larger proportion of cardiac affections than was met with in the series of dispensary cases. The details of the cardiac affection may be stated as follows:

Systolic murmur at apex, - - - -	10 cases.
Systolic murmur at apex and pulmonic area, -	1 case.
Diastolic murmur at pulmonic area, - -	1 „
Irregularity of cardiac sounds, - - -	2 cases.
Pericarditis, - - - - -	1 case.
Total, - - -	<hr/> 15 cases.

In 5 of the cases distinct increase of the cardiac area to the left was also noted, and in 7 no increase of the cardiac area could be made out.

In the hospital cases we have a proportion of exactly 60 per cent. in which there was unmistakable valvular affection of the heart associated with the chorea, as compared with 31 per cent. of the dispensary cases. This may be taken as evidence of the greater severity of the affection in patients admitted into the wards. If we take the average of both series of cases we get a percentage of 45 in which cardiac disease was associated with the chorea, a sufficiently large proportion to establish a very close clinical and etiological relationship between the two conditions.

(9) *Condition of the Urine.*

The urine was found to be normal in 20 cases, and abnormal in 1. As regards 4 of the cases there was no information.

In the series of ward cases albumen was only discovered in 1, that of the young girl who was found to be pregnant. In her case a few granular tube-casts were also detected. On her second admission, the chorea having recurred with the advent of her second pregnancy, there was no albumen in the urine, which was normal in all respects.

In 20 of the ward cases a note of the specific gravity of the urine was preserved. In 11 of these cases only one observation of the specific gravity of the urine was recorded, but in 9 of them a series of consecutive daily observations, varying from 20 to 54 days, was made.

In the 11 cases where the specific gravity was noted on only 1 occasion the average density was 1022, the maximum being 1032, the minimum 1015.

Of the 9 cases in which a series of daily observations of the specific gravity was made, the following are the particulars:

1. Average of 20 daily observations, - - 1016.
 Maximum, 1023.
 Minimum, 1010.
2. Average of 23 daily observations, - - 1013.
 Maximum, 1018.
 Minimum, 1010.
3. Average of 23 daily observations, - - 1017.
 Maximum, 1024.
 Minimum, 1011.
4. Average of 28 daily observations, - - 1022.
 Maximum, 1039.
 Minimum, 1015.
5. Average of 25 daily observations, - - 1022.
 Maximum, 1030.
 Minimum, 1014.
6. Average of 33 daily observations, - - 1021.
 Average of first 16 observations, 1026.
 Average of second 17 observations, 1017.
7. Average of 42 daily observations, - " 1014.
 Average of first 21 observations, 1015.
 Average of second 21 observations, 1012.
 (2nd admission, 54 observations, 1015.)
8. Average of 28 daily observations, - - 1013.
9. Average of 25 daily observations, - - 1025.

The average specific gravity of the urine in this series of 9 cases in which extended daily observations were made is 1016, a result which clearly shows that the specific gravity

of the urine in chorea is on the whole rather below than above the normal point.

The reaction of the urine was noted in 20 out of the 25 ward cases, and in all of them it was found to be acid.

Here again, then, as in the dispensary cases, it is seen that there is no special derangement of the renal function associated with chorea.

(10) *Distribution and Severity of the Movements.*

The following table shows the distribution of the choreic movements :

General,	-	-	-	-	-	-	-	16 cases.
Right-sided (chiefly),	-	-	-	-	-	-	-	5 „
Left-sided (chiefly),	-	-	-	-	-	-	-	4 „
								<hr/>
Total,	-	-	-	-	-	-	-	25 cases.

As regards the severity of the choreic movements the hospital cases were classified as slight and severe.

Movements slight,	-	-	-	-	-	-	-	10 cases.
Movements severe,	-	-	-	-	-	-	-	15 „
								<hr/>
Total,	-	-	-	-	-	-	-	25 cases.

Here, again, the table shows that, as compared with the dispensary series, the cases admitted into the wards are altogether of a more severe type.

(11) *Time under Observation.*

In cases admitted to the wards it is much easier to obtain accurate information as to the length of time the patients were under observation, and this is shown in the following table :

One month and under,	-	-	-	-	-	-	-	9 cases.
Two months	„	-	-	-	-	-	-	11 „
Three months	„	-	-	-	-	-	-	3 „
Four months	„	-	-	-	-	-	-	2 „
								<hr/>
Total,	-	-	-	-	-	-	-	25 cases.

Of cases that were admitted to the wards on more than

one occasion, the following table shows the time the patients were under observation:

Two months and under,	-	-	-	-	3 cases.
Three ,, ,,	-	-	-	-	1 case.
Total,					<hr/> 4 cases.

The first of these tables may be compared with the first given under Section (4) of the present series, when it will be seen that on the whole the treatment in hospital tends to shorten the duration of the attacks, although no absolute comparison of the two tables can be drawn.

It is not my intention to comment further upon the facts which have been tabulated in the foregoing pages. My object has simply been to show in a statistical form the frequency with which the well-recognized clinical phenomena of the disease occur in a fairly large series of consecutive cases. On the whole it may be admitted that the tabulated results of my personal experience confirm the teaching of other observers with regard to the clinical history of chorea.

It is generally taught that psychical disturbance, more or less severe, is a frequent accompaniment of chorea. My records upon this point contain no information that could have been made use of in the present inquiry. Certainly in my dispensary cases mental symptoms never prominently obtruded themselves; and even in some of my most severe cases observed in the wards the mind remained perfectly clear throughout. I do not wish it to be understood that in any sense I am asserting that mental symptoms do not occur in chorea, because in all probability, had the point been more definitely inquired into, a considerable number of cases presenting psychical phenomena might have been discovered. A case of *chorea insaniens* I have never seen; and my experience of chorea leads me to agree entirely with the latter part of the following statement, quoted from Osler: "Psychical disturbance is rarely absent in chorea; fortunately in the majority of cases it is slight in degree."

When this paper was commenced I had intended to include the subject of treatment, but this must be left for a future contribution.

SOME CONSIDERATIONS REGARDING THE THERAPEUTICAL ACTION OF IODINE AND IODIDES.

By RALPH STOCKMAN, M.D., F.R.C.P.E.

THE therapeutical history of iodine does not extend over a very long period of years. The element was only discovered in 1812, and was first introduced into medicine in 1820 by Coindet of Geneva, when, in a short paper entitled *Découverte d'un nouveau remède contre le goître*,¹ he drew general attention to the efficacy of iodine and the iodides of potassium and sodium in causing the disappearance of soft bronchoceles. But the use of iodine in goitre, although in much less convenient form, dates back to a very remote period. The ash of bladderwrack (*Fucus vesiculosus*), and burnt sponge, were well-known remedies at the time, and, in searching for the active constituent common to both, Coindet came to the conclusion that it must be iodine, a conjecture which his therapeutical observations very soon showed to be correct. The knowledge of the value of burnt sponge in goitre and scrofula had been widely spread by Arnaud de Villeneuve (thirteenth century), but it had long previously been in less general use in Europe, while in China and among the natives of South America marine algae and certain iodine waters seem to have been employed in similar cases from prehistoric times.

Coindet's discovery gave a great impetus to the use of iodine and its compounds in medicine; but notwithstanding the many researches which have since been made into their exact mode of action, this still remains obscure in many important particulars, most writers escaping the difficulties by

classing them vaguely as "alteratives." The ordinary methods of pharmacological investigation, such as the examination of the circulation, the nervous system, blood, etc. in animals under the influence of iodides, have conspicuously failed to explain satisfactorily many of the reasons of their undoubted therapeutical activity. On the other hand, the knowledge which we have now obtained of the functions of the thyroid gland, of the absolute necessity of its secretion for healthy existence, and the fact that Baumann and Roos² have proved that an iodine-containing organic compound is the active constituent of the secretion, afford a probable explanation of certain of the actions of iodine in diseased conditions, and of some at least of its occasional deleterious effects.

We may begin with goitre in which iodine is the remedy *par excellence*. Coindet and others soon found that while most cases of goitre rapidly improved under iodine, yet a few of them developed very alarming symptoms, and some deaths were recorded. Nearly all contemporary writers attributed these results to the direct poisonous action of the iodine, and the remedy came to be regarded with much distrust. The symptoms varied only in degree, and consisted in great and often alarming emaciation, atrophy of the breasts or testicles, rapid heart-action, palpitation, tremors, nervousness and sleeplessness. These cases were not uncommon in Switzerland, and occurred with such small doses as $\frac{1}{100}$ to $\frac{1}{30}$ grain iodine, or $\frac{1}{24}$ grain potassium iodide daily. It is quite clear now that these symptoms are due to thyroid poisoning, an increased secretion, or increased discharge of the secretion into the blood, being evidently provoked by the administration of iodine. I have lately seen exactly similar symptoms occur in a lady, aged 36, during the cure of a goitre for which no iodine was given. She had a large soft goitre which was causing absolutely no symptoms except a little discomfort in swallowing. On her going to the country for change of air, the goitre began to diminish rapidly in size, and she suffered from emaciation, nervous excitement, tremors, excessive palpitation, and insomnia. The goitre completely disappeared in about two months, the symptoms persisting for three months

longer, after which she gradually recovered. Here, iodine played no part whatever in producing the symptoms. Prévost, Lebert,³ and Röser⁴ in discussing these cases had, at different times, expressed the opinion that the condition might be due to poisonous products from the breaking-down goitre; but as nothing was then known regarding the function of the thyroid, no attention was paid to their views, the iodine was universally regarded as the cause, and the condition received the name of "iodism" or "iodic cachexia." It has been often seen in goitrous dogs to which iodine has been given, and many cases have been recorded by Rilliet,⁵ in some of which burnt sponge, or small doses of iodine-containing mineral waters, and even living at the seaside, have induced it in goitrous men or women.

It is a difficult matter to decide whether these symptoms ever occur in non-goitrous persons on the administration of iodine. Even the existence of this form of "iodism" has been denied. Ricord and Velpeau in Paris, out of 15,000 cases⁶ in which iodine or potassium iodide had been given, never saw an instance of it, and Hermann⁷ in Vienna, with 50,000 cases, was in a similar position. It is certainly therefore very rare, but, in a few cases, loss of subcutaneous fat and atrophy of the mammae or testicles have apparently followed directly on the use of iodine and iodides. Most writers of text-books seem to regard atrophy of glands as a common effect of iodides, but this is not my opinion, and on consulting the original records of reported cases I find it usually stated that the persons who suffered in this way were goitrous. This most important element in the case has generally been omitted in the citation or abstract. But it seems quite clear that, when these effects do occur, they are not primarily due to iodine or iodides, but to an increased secretion of thyroid juice provoked by the drug. A more careful examination of these cases might have revealed an enlarged thyroid gland.

Owing to the rarity of this form of "iodism" in all countries in which goitre is not endemic, it is very little known, and hence the more familiar form of poisoning has gradually come to be labelled "iodism." The symptoms of it are very familiar, and

arise from irritation of the nasal, buccal, and respiratory mucous membranes, accompanied by skin eruptions, and a very considerable feeling of depression. Common as it is, we have no exact knowledge of the conditions under which it occurs. It is generally assumed that the irritation is due to free iodine, but the element has never been detected "free" in any case. It sometimes develops on one side only of the nasal mucous membrane, and hence the conditions which induce it must be local. It has been suggested that the iodides are decomposed by ozone,⁸ by carbonic acid,⁹ by nitrites,¹⁰ and by the lymph cells,¹¹ but none of the theories advanced stand the test of experimental criticism. Numerous methods of preventing its occurrence have been suggested. Ehrlich and Kroenig¹² advise large doses of sulphanilic acid upon the theory that it will combine with the nitrites present and prevent them decomposing the iodides. It is doubtful if this union could take place *in corpore*, and the suggestion resolves itself into a theoretical remedy to counteract a still more theoretical cause of the condition. Quinine,¹³ bromides,¹⁴ potassium bicarbonate,¹⁵ and sarsaparilla¹⁶ have all been advanced as preventives, while arsenic and strict cleanliness have been said to lessen the tendency to skin eruptions. I may say that I have frequently tried all these so-called preventives of iodism except sulphanilic acid, but have never seen any benefit from them. A statement is frequently made that by doubling or increasing the dose of iodide the coryza and other symptoms may be cut short. My own experience is very distinctly contrary to this, as I find that either stopping the medicine or lessening the dose usually causes a rapid abatement of the symptoms, while an increase of dose tends to aggravate them. On searching through the literature of the subject, I have been unable to find any authority for the statement that an increase of the dose cuts short the attack.

In aneurism the value of potassium iodide is now well established. The explanation given of its beneficial action has usually had reference to a supposed depressing effect on the heart, with dilatation of the small arteries, and lowering of blood pressure. But experiments have shown that no such

actions can be rightly attributed to it. Iodine and sodium iodide have only a very trifling action on the heart and circulation, while potassium iodide owes its effects in this direction to its potassium component, and as long as it is given in only moderately large doses it tends to strengthen the heart's beat and raise the blood pressure, rather than to depress them.

In atheroma, also, iodides have in many cases, even in those which are not syphilitic, a retardative and sometimes, apparently, a curative effect, no explanation of which has been forthcoming. It is probable, however, that in some of these cases early atheroma and aneurism may be due to a lessening of the thyroid secretion, and that the iodide has a counteracting effect on this. Vermehren has pointed out that premature senility and myxoedema have many features in common. The change in the expression, the falling out of hair and teeth, the dryness of skin, the dulling of the nervous system, the decreased metabolism, the low temperature, and sensitiveness to cold, all strike one as features common to both. The changes in the arteries and capillaries (increase of connective tissue and its consequences) are also common to both conditions. There must be many factors in the changes consequent on old age, but it is highly probable at least that a degree of failure of the thyroid secretion is one of them. This seems also to have occurred to Horsley, who remarks that in his opinion an active thyroid gland is a necessity for maintaining a vigorous old age. If this be so, the increased secretion of thyroid juice under the action of iodides may lead to an absorption of the atheromatous tissue, such as occurs in myxoedema, but of course on a much lesser scale.

In tertiary syphilis, also, it seems highly probable that absorption of new growths is not so much due to a primary iodine action as to some stimulating effect of the drug on thyroid activity. The prevailing theory of the action of potassium iodide in syphilis is that the salt is broken up by some unknown agency in the new growths, that the free iodine combines with and breaks down the albumin

of the cells, which then die and become absorbed. Hence the prevailing idea that the larger the dose the better the action. Such a chemical reaction as has just been described is, to say the least, most improbable, and there are, besides, certain facts which militate against this view. Coindet, Mayo, and others cured syphilis with very small doses of iodine, and iodide of potassium was at first given in $\frac{1}{2}$ to 2 grain doses. Iodoform, iodothylin, and ferrous iodide have been successfully used in syphilis in very small dose, although not so effectively perhaps as larger doses of the alkaline iodides. Potassium bromide is absolutely ineffective in syphilis, and therefore the cure of tertiary growths is an iodine action, and is quite marked with any dose. The most probable explanation is that the iodine acts in these cases by bringing about an increased secretion of the powerfully resolvent thyroid juice, and we know that small doses are capable of doing this under certain diseased conditions. Healthy persons can take very large quantities of potassium iodide¹⁷ (up to 600 grains per day or even more) without suffering any ill effects, except perhaps thirst and some slight catarrh, hence it seems probable that the salt passes out of the body without any of its iodine being utilized by the thyroid or other cells. In short, it has in health no more action than a similar amount of sodium chloride would have, that is to say, only a purely saline action. In disease it is probably utilized by the thyroid gland, and hence its different effects. We have an analogous state of affairs in the case of iron. In health, when the haemoglobin and corpuscles are normal, the administration of iron does not increase either, nor has it any apparent action on them, but as soon as anaemia occurs the utilization of the iron is immediate and its effects most marked.

In lead and mercurial poisoning it is probable that alkaline iodides act simply as salts, and have not any specific iodine or thyroid action. The discovery of their efficacy in these cases is due to Melsens.¹⁸ In hardening tissues with mercuric chloride for microscopic examination,

he removed the metal with potassium iodide, and on his recommendation it was successfully used in mercury poisoning in man. Shortly after, it was also very successfully tried in lead poisoning. Bromide of potassium is stated to be quite as effective in these cases, but I myself have no experience of it. Both of them most probably owe their efficacy simply to their chemical properties of being able to dissolve up albuminous compounds of lead and mercury, which then become capable of excretion by the kidney.

Such considerations are to a certain extent theoretical, and must remain so until more systematic clinical investigation has been made into the action of iodides; but few drugs are used in more haphazard fashion, and until we obtain more precise information regarding their mode of action and the conditions under which they act, it is hopeless to expect any advance in precision in their practical clinical application.

REFERENCES.

- ¹Bibliothèque Universelle, xiv., 1820.
- ²Ztschr. f. physiol. Chemie, 1895.
- ³Krankheiten der Schilddrüse, 1862.
- ⁴Archiv f. physiol. Heilkunde, 1848 and 1859.
- ⁵Mémoire sur l'iodisme constitutionnel, 1860.
- ⁶Bull. de l'Acad. de Méd., xxv., 1859.
- ⁷Oesterreich. Ztschr. f. pract. Heilkunde, 1861.
- ⁸Buchheim, Archiv f. expt. Pathologie, iii., 1875.
- ⁹Kämmerer, Virchow's Archiv, lix., 1874.
- ¹⁰Sartisson, Thesis, Dorpat, 1866.
- ¹¹Biuz, Lectures on Pharmacology.
- ¹²Charité Annalen, x., 1883.
- ¹³Wallace, Lancet, 1835-36.
- ¹⁴Norris, Amer. Med. News, 1885.
- ¹⁵Fenwick, Lancet, 1875, ii., 698.
- ¹⁶Hill, Lancet, 1876, i., 594.
- ¹⁷Puche, Gaz. des Hôp., 1842. Sisson, Lancet, 1860, i., 382.
- ¹⁸Comptes Rendus, 1844, xviii., 522.

A CASE OF EXTENSIVE CIRSOID ANEURYSM OF THE SCALP.

By A. ERNEST MAYLARD, M.B., B.S., LOND.,
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THIS case seems of peculiar interest in that while occurring in a region where "aneurysm by anastomosis" is most commonly met with, the tumour was of unusually wide distribution, involving nearly the whole scalp.

EXTRACT OF REPORT TAKEN BY DR. JAMES M'HAFFIE.

T. M., aged 32 years, was a patient of Dr. Tawse, of Whitehaven, Cumberland, by whom he was sent into the Victoria Infirmary. About nine years ago he noticed a small red "warm" patch on the back of his head. At this time it caused him neither pain nor inconvenience, and his attention was not particularly directed to it until his haircutter told him that he had a tumour on his head. More recently it had become a distinct source of trouble to him, causing at times shooting pains and a throbbing sensation. Three weeks ago he felt a slight roughness on the surface of the tumour, and in attempting to scratch it off with his finger nail a quantity of blood suddenly escaped. He is unable to give any distinct history as to the cause of the tumour except that he states he once injured the back of his head with a nail.

After shaving the head the following conditions were found. Over the region of the posterior fontanelle there exists a soft, slightly pulsatile swelling, projecting for about half an inch above the surrounding surface of the scalp, and extending

over an area two and a half inches in diameter. Towards its periphery it shelves off into the neighbouring tissues except where it is connected with dilated vessels. On the surface of the tumour the skin is in places exceedingly thin, and appears as if at any time it might give way. At one spot is seen the cicatrix of the recent rupture. Pulsation is very marked. Pressure empties the tumour to a considerable extent.

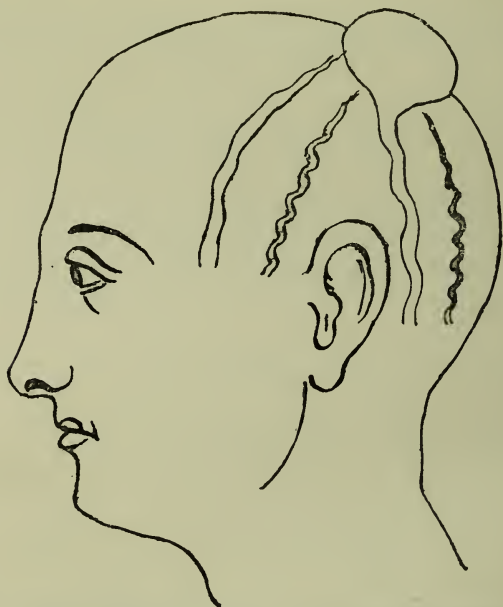
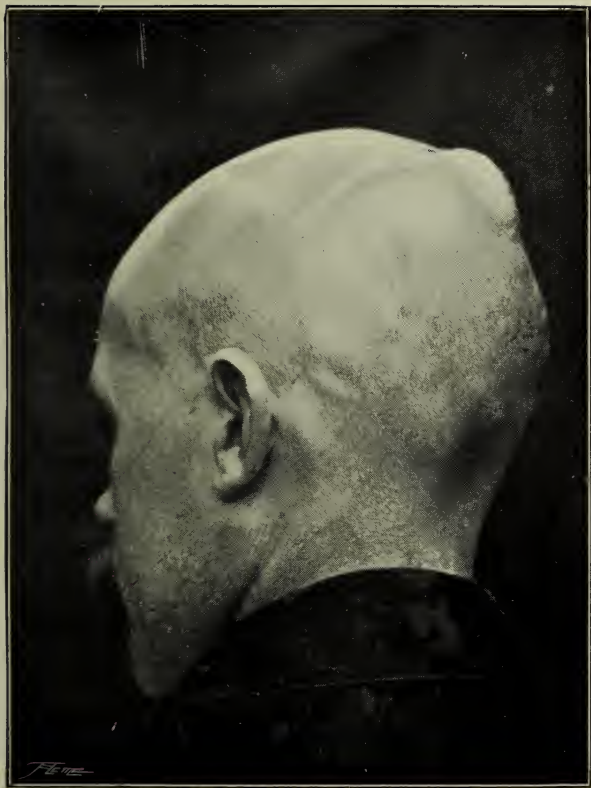


FIG. 1.—Cirroid aneurysm of the scalp. Diagrammatic representation of the tortuous and dilated vessels passing to the tumour.

On examining carefully the other parts of the scalp, numerous dilated and tortuous vessels are seen passing upwards from the basal portion of the vault towards the tumour. Some can be plainly traced into the growth, while others disappear short of reaching it. The common feature of all these vessels is their tortuosity, more marked, however, in the arteries than in the veins. These enlarged vessels appear to be the normal vascular channels of the scalp. Thus, on the left side of the head (see Fig. 1), both temporal vein and artery are involved; on the right side (see Fig. 2) also the temporal



Cirroid aneurysm of the scalp. (From a photograph by Dr. Watt.)

vessels, but in addition the supra-orbital vein and the posterior auricular vein. This last proves the largest connecting branch of the growth, so large and tortuous indeed is it, as it passes from the tumour, that it seems as if it formed a true prolongation outwards of it. On the occiput all four occipital vessels are involved, and from the right occipital artery a distinctly tortuous and pulsating branch passes upwards and outwards.



FIG. 2.—Cirsoid aneurysm of the scalp. Diagrammatic representation of the tortuous and dilated vessels passing to the tumour.

Remarks.—Notwithstanding these numerous enlarged and visibly pulsating vessels, the man never seemed to have been conscious of them, the existence of the tumour alone troubled him. Indeed it was only on shaving the head that he became aware of their existence. We found that digital pressure applied to the arteries at their basal origin completely stopped all pulsation in the tumour, and it was this effect that led me to consider the advisability of attempting to excise the growth rather than try any less radical measures.

The first stage of the operation was performed on Novem-

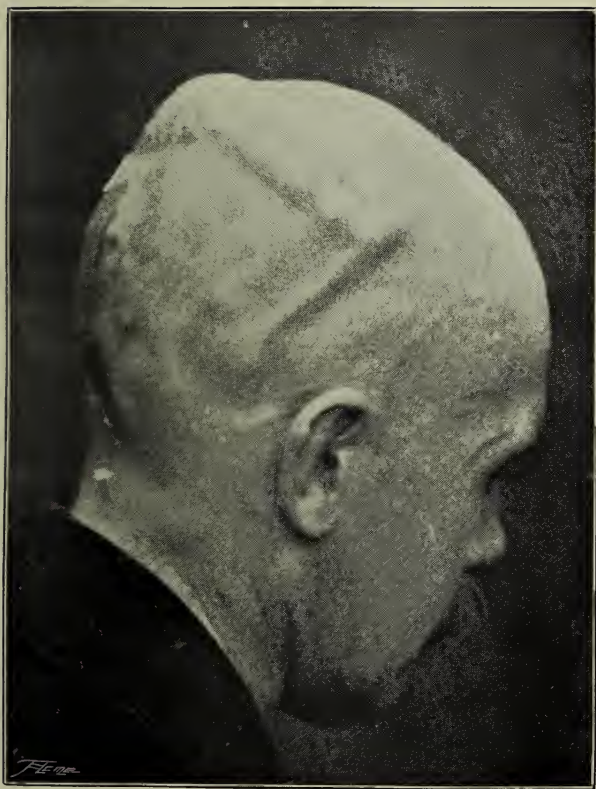
ber 25th, and consisted in applying a ligature to all the tortuous arteries as low as possible. So vascular appeared to be all the scalp cellular tissue, that each incision, though small, to secure the artery, involved a good deal of bleeding. When the ligatures had all been applied it was found that pulsation had completely stopped in the tumour.

Three days later, on the 28th, the wounds were examined and found practically closed, but pulsation was recognizable in the tumour. On December 5th the wounds were quite healed and stitches all removed; pulsation of the tumour, however, was more marked.

On December 9th the second and final stage of the operation was performed; this consisted in excision of the tumour by the following practically bloodless method:

It was decided to remove it by a diamond-shaped incision for two reasons—(1) that each side of the diamond might be bloodlessly completed before attempting another; and (2) that, after the growth was excised, the gaping wound would better lend itself to partial closure by approximating the sides of the diamond.

Before making the first incision, acupressure needles were passed at a distance of about half an inch from the proposed line of incision. It was thus possible to strangulate all the vessels on the distal side, and render that edge of the incision bloodless. A cut was rapidly made right down to the bone, and the tumour edge of the wound at once clamped with forcipressure forceps. The most bleeding, and that not much, occurred at this first incision, because the want of separation of the tumour, which was allowed by the subsequent cuts, did not admit of easy and rapid application of the clamp forceps. The remaining three sides of the diamond were cut in the same way, and when the tumour was finally removed we had a bloodless wound surrounded by some eight or ten acupressure needles. It then only remained to withdraw one needle at a time, and in doing so to secure each bleeding vessel as it showed itself. A stitch or two was passed between the edges of the angle, and thus the raw surface was somewhat reduced in size.



Cirroid aneurysm of the scalp. (From a photograph by Dr. Watt.)

There is little to add regarding the further progress of the case. The wound healed rapidly and well; and the patient left the Infirmary on the last day of December cured of his complaint. There existed no indication of any dilated or pulsating vessels in the scalp.

It may seem that I have entered with undue prolixity into the description of the operation performed. But when it is remembered that fatal results from excessive haemorrhage have more than once occurred in the attempt to excise tumours of this nature from the scalp, it would not have been right in describing the operation to omit any step or precaution taken in its performance. It is quite possible that I might have successfully excised the tumour at the same operation as that in which I applied ligatures to all the different vessels. But I must own to the somewhat delusive hope which I had, after securing these vessels, that the complete cessation of pulsation which followed might end in cure. Against, however, any such possibility was the past record of numerous failures which had followed similar attempts. The treatment by electrolysis which we discussed was abandoned in the belief that careful incision would prove a much more rapid and certain way of dealing with the tumour. The practically bloodless nature of the operation was entirely due to the previous insertion of the acupressure needles and the way in which all bleeding points were secured as one needle after another was removed separately.

[The photographs accompanying this paper were kindly taken for me by Dr. Watt.]

ON THE DIAGNOSIS AND TREATMENT OF A CASE OF PATENT URACHUS.

By WILLIAM L. REID, M.D., F.F.P.S.G.,

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Maternity Hospital.

THE following case is published, partly because of the difficulty of diagnosis, and partly because of the difference of opinion in regard to the proper method of treatment of such cases. On the surface, it looks as if the treatment should be simple enough, when the diagnosis has once been made.

What follows in inverted commas is copied from the Ward Journal at the Western Infirmary, as written by Dr. James D. Cochran, the then House Physician. The rest is my own remarks and opinions on the symptoms and progress of the case.

"Mrs. M., *aet.* 25. Admitted November 12, 1897. Married three and a half years ago, and since then has had three children. Twins at full time and normally, a year after marriage; and the third child in July last, about full time and normally. Since the birth of the twins patient has complained of pain in the right ovarian region, which the doctor thought due to an enlarged ovary, and for which patient had electricity used and a few blisters, without any good result. In April of this year pain began in the region of the umbilicus, and a fortnight afterwards swelling showed itself and pus was discharged; and this has continued with occasional intervals of a week or so until now. No leucorrhœa; menstruation and defecation normal.

"A very fine probe passes through some granulation tissue in the depression of the umbilicus downwards and backwards for three inches, that being the whole length of the probe; a No. 4 sound cannot be made to enter the sinus.

"P. V. cervix is high, split on the left side, eroded, but not ectopic. There is no evidence of a pelvic cellulitis, but the sound, passed easily to three inches, showed that the fundus, not quite fixed, was yet greatly restricted in its movements."

The above note is, I think, defective, inasmuch as it fails to state that the sound passed towards the right side as well as downwards and backwards; and this gave countenance to the belief expressed by one of the surgeons to the hospital, as well as by two or three other medical men who examined the case, that it was one of abscess in the region of the right ovary, and in which pus had found its way to the surface in this unusual locality.

It was decided to examine the patient under chloroform.

"November 24th. To-day, under chloroform, Dr. Reid passed a long thin probe into the sinus opening at the umbilicus, and this, passing quite easily downwards along the abdominal wall, finally appeared externally at the urinary meatus."

I am afraid this is not strictly true. The instrument used was what is commonly called a post-mortem probe, 10 inches long, and it passed downwards with many hitches until its eye reached the umbilicus. A peculiarity, afterwards explained, was that the probe passed backwards as well as downwards, so that, near the pubis, it was so deep that it could not be felt through the anterior abdominal wall.

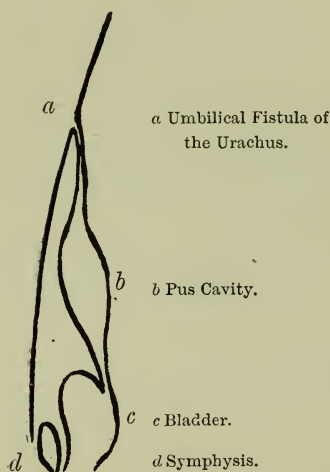
I thought it had passed into the vagina, but, on examination, I was astonished to find it projecting from the urethra. Then, for the first time, I recognized that I was dealing with a case of patent urachus.

So far as I can find, there is no case on record in which the diagnosis was made by passing a sound from the umbilicus to the urethral orifice. Dr. Freer (*Annals of Surgery*, Vol. V., St. Louis, 1887) mentions a case where the diagnosis was made

by the injection of starch into the urachus and its detection in the urine by the iodine test. In others, urea was found in the fluid obtained from the sinus.

The question now arose as to the best method of dealing with the affection. I consulted various surgical friends. One said, nothing short of cutting out a longitudinal strip of the

whole anterior abdominal wall would be safe or satisfactory. Another, that it would suffice to open the urachus from umbilicus to bladder and cause it to granulate up. The former plan, I thought, would lead to considerable risk of septic peritonitis; the latter would leave a very weak abdominal wall, as the sinus was deep, and so lead to a great after-risk of hernia. It was decided to try cutting off the communication with the bladder and drainage of the abscess cavity.



“December 2nd. After some trouble, and of course under chloroform, the probe was passed to near the bladder and was cut down upon about two inches above the pubis. Before reaching the probe, a layer of apparently inflammatory tissue like wax-cloth was cut through and below this a vascular membrane like the mucous membrane of the bladder. This bled freely and gave trouble. It was evident that a cyst had been entered, the cavity being one inch broad and one inch and a half long. Below this, the probe was found in a narrow channel, and in the attempt to reach it through the anterior abdominal wall, the bladder was opened, and it was afterwards found that the urachus ran into the bladder behind, instead of at the summit. About one inch of the lower part of the urachus was destroyed by the actual cautery. The bladder was closed by a catgut suture. A bit of iodoform gauze was carried from the wound to the umbilical opening

by means of a silk thread in a surgical probe and the rest of the wound closed with catgut sutures, the upper pair of which were left untied, to admit of drainage.

"The cyst of the urachus was not dissected out because it would have involved opening the peritoneal cavity; and in the presence of pus and urine this was considered dangerous."

This is a fair report of the operation. I did not expect to open into the general cavity of the bladder before reaching the lower part of the urachus, having the idea that it would enter at the very highest part of the organ. The part of the urachus near the bladder seemed to have nothing behind it but peritoneum, so that it could not have been dissected out without opening the peritoneal cavity. In the Journal the fact is not mentioned that the urachus near the bladder was most carefully sutured with a fine needle and catgut.

"December 4th. Since the operation the patient has done very well. The urine has been drawn off by catheter every two hours. For the first day it contained blood and a large amount of pus, but within the last twenty-four hours it has become quite clear and entirely free from pus and blood.

"The temperature, after the operation, rose to 99.6° , but has not exceeded this; and, to-day, has fallen to normal. The wound is dressed to-day and no appearance of inflammation is observed. The patient feels very comfortable. The catheter is now to be used only every four hours.

"December 7th. To-day the case is again dressed, and the two uppermost sutures tied after withdrawal of the drain of iodoform gauze, in the place of which a thread of silk is left. The temperature has been normal since last note, and the wound shows no appearance of inflammatory action. The urine, since the 5th, has been passed voluntarily, without discomfort or difficulty, and remains clear, without pus or blood.

"December 14th. To-day the stitches are removed, and the silk thread passed from the upper part of the wound to the umbilicus withdrawn, causing slight bleeding from the latter, but not bringing with it any pus. Patient is to be allowed up to-night. She feels very well in every respect.

In particular she has no pain in the right ovarian region. The urine gives her no trouble.

"December 16th. To-day the wound is again looked at. There is a slight appearance of moisture at the umbilicus, which may be either perspiration or a slight serous discharge."

The slight discharge just mentioned was not seen again, and four days afterwards the patient was discharged, and, when heard of last (July 25th, 1899), had continued quite well.

There seems no reasonable doubt but that the patent condition of the urachus existed in this woman from her birth. What brought it into prominence? May it not have been over-distension of the abdominal wall by the carriage of twins to the full time? It will be noted that in the first part of the Journal note it is stated that, since the birth of the twins, she had complained of pain in the right side, which was considered to be ovarian, and treated by electricity and blisters; and it was in this connection that she was admitted to one of my beds.

As regards the operation, the question with me was, whether the bladder could be shut off and the sinus destroyed without opening the abdominal cavity. Of two cases mentioned by Freer, one was met in making an abdominal section, and, although the point of exit from the bladder was carefully sutured with catgut, the patient died of peritonitis in a few days. The other also died of peritonitis, although the peritoneum had not been opened, but simply a flesh flap formed to cover the orifice of the urachus.

In the present case the wound was completely healed and the patient free from any symptom of disease fourteen days after the operation, and the cure has been permanent.

A CASE OF GASTRIC TETANY, WITH AN ACCOUNT OF THE MICROSCOPIC APPEARANCES FOUND IN THE MEDULLA AND SPINAL CORD.

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Children; Foulis Memorial Scholar.

From Dr. Lindsay Steven's Wards in the Glasgow Royal Infirmary.

TETANY in the adult is a somewhat uncommon disease, and a case of tetany ending fatally is distinctly rare. The fatal cases, when they do occur, seem to be chiefly cases of what has been called "gastric tetany," meaning by that cases in which the tetany-convulsions have been associated with a chronic gastric disorder. Not more than 50 such cases have, so far as I know, yet been put on record, and the mortality among these is said to have been as high as 75 per cent.

I do not, however, propose to enter here on any discussion as to the nature of gastric tetany, as the subject has recently been very ably reviewed by Dr. Trevelyan in the *Lancet* for 24th September, 1898, and those wishing a general survey of the subject I would refer to that paper. But I wish to put on record what seemed to be almost undoubtedly another case of this disease, and I shall also take the opportunity to describe the microscopic appearances found at the post-mortem examination in the medulla and spinal cord.

The history of the case is, unfortunately, rather scanty, owing in part to the patient's mental condition while under

our observation, and in part to the fact that we could find no relation or friend of the patient who seemed to know any particulars of either her personal or her family history. As to its being a case of tetany there can be little doubt, for the convulsive movements were characteristic of those seen in that disease. We are, however, without any definite knowledge as to the condition during life of the patient's digestive apparatus. But the post-mortem appearances of the stomach strongly suggested a gastric disturbance of considerable duration, and the history of headaches and of being "somewhat addicted to drink, though not recently," may also be taken as evidence in favour of that view.

The condition of the kidneys found post mortem made one consider the possibility of the convulsions being uraemic in origin. Diseased kidneys, by preventing elimination of poisonous material, might quite well determine the onset of convulsive movements. But then the convulsions in this case were so characteristically those of tetany that we should not be justified in regarding them as entirely due to uraemia. Moreover, Dr. Trevelyan, in the paper already mentioned, draws special attention to the fact that it is not at all uncommon to find nephritis associated with cases of gastric tetany. The possibility of the case being one of antipyrin or antifebrin poisoning seems very remote, for I am informed on good authority that neither of these drugs has ever been known to produce such symptoms as seen in this case.

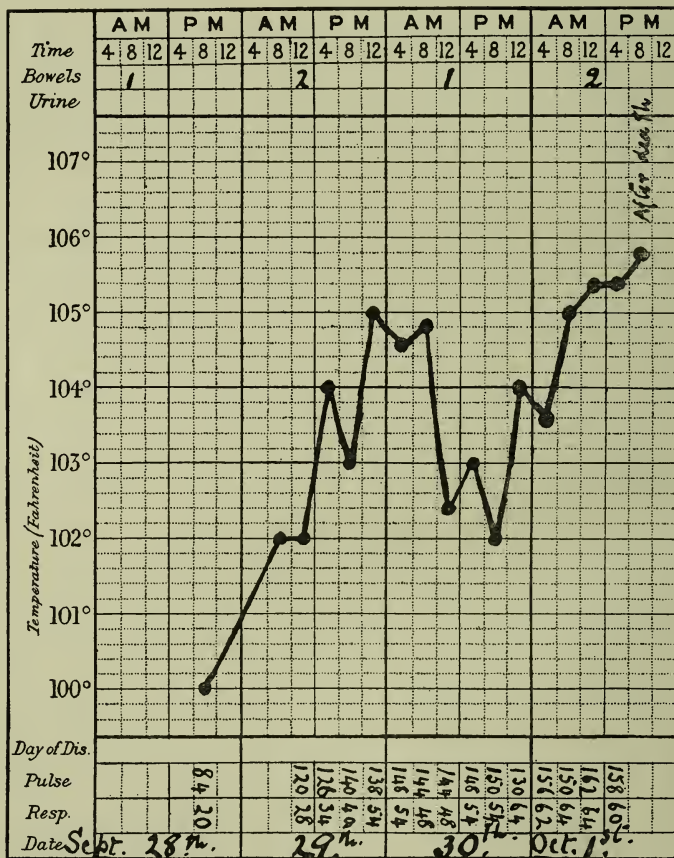
I. D., aet. 41, a domestic servant, was admitted to ward viii. of the Glasgow Royal Infirmary on 28th September, 1898, at 6 P.M., said to be suffering from some unknown form of poisoning. She had been in the habit of taking powders for the relief of headache, and two of these powders were brought to the infirmary by her friends. There is some doubt as to their nature, but the chemist in Arbroath from whom she had obtained powders since 1892, reports that they frequently supplied her with antipyrin, and that on subjecting one of the two powders to chemical tests they found it to consist of ten grains of antifebrin, which they say she must have obtained in Glasgow.

She had been in Glasgow for a fortnight before her admission, looking for a situation. On the morning of the 24th September she vomited and complained of headache. The headache continuing, she took a powder on the morning of the 25th. On the evening of the 26th, as she was still feeling giddy and unwell, she took a second powder. The next day she was delirious, and in the evening she was thought to be unconscious, and a doctor was sent for. He did not, however, see the patient until the 28th, and having heard the story of the powders, and regarding the symptoms as perhaps indicative of some form of poisoning, he ordered her removal to hospital.

On admission patient was first regarded as being a case of poisoning, and she was treated accordingly. But more careful observation of the symptoms, cast much doubt on this diagnosis, and pointed rather to some definite organic disease. The patient lay for the most part with the eyes wide open and staring, the pupils being of medium size, and responding to light and accommodation. There was spasmodic contraction, with occasional twitchings and tremors, in the muscles of the face, arms, and legs. The lips were compressed, and in a state of constant tremor. There were also convulsive twitchings in the other facial muscles, and a very characteristic *risus sardonicus*, coming and going, fully exposing the teeth, and importing a distinctly smiling expression to the profile view of the face. A marked trismus was also present. The irritability of the facial muscles and their nerves was much increased, and percussion over the facial nerve, especially on the left side, produced a contraction of all the muscles on that side of the face.

There was marked spasm in both arms, these being adducted at the shoulders, flexed at the elbows and slightly flexed at the wrists. The hands assumed the attitude characteristic of tetany. There was flexion at the metacarpo-phalangeal joints, with extension of the phalanges, adduction of the thumbs and contraction of the thenar and hypothenar muscles. The irritability of these arm muscles was also greatly increased, any attempt at straightening the arms

leading to violent muscular contractions, which also spread to the muscles of the front of the chest. Twitchings were also present in the toes. The toes were flexed and the feet extended and inverted in the position of talipes equino-varus. The electrical reactions were not tested.



As to patient's mental condition, she seemed only partially to understand what was said to her. She attempted to reply to questions, but her answers were rather disjointed statements and not easily made out. During the first night of her residence in the ward she was very restless, lying with her eyes wide open, picking at the bed-clothes and

frequently talking to herself. Indeed her appearance was that of a person in the incipient stage of delirium tremens. The next day patient passed into a state of stupor, and this gradually increased till some twenty-four hours before death, when the coma became complete, and patient had to be fed by the nasal tube. As coma deepened the twitchings lessened, though the rigidity of the muscles still continued.

No definite information could be obtained in regard to sensation.

Examination of the other systems of the body revealed little worthy of note. There was no *tache cérébrale*, and no retraction of the abdomen. There was no rash, and the only abnormality of the skin was the presence of one or two brown stains on the abdomen. The heart, lungs, liver, and spleen seemed quite normal. The urine and faeces were passed in bed, the motions being loose, but without any abnormal appearances. A specimen of urine drawn off by catheter had a specific gravity of 1026, and it contained a small amount of albumen. The temperature, as will be seen on the chart, began to rise on the evening of admission, and it ran a high course. The pulse, too, became very rapid. The respirations were noted to be somewhat irregular and sighing, but never definitely to assume the Cheyne-Stokes character.

The patient died on 1st October, exactly three days after admission.

At the post-mortem examination the body was noted to be well developed, though somewhat emaciated. The pericardium contained about half an ounce of clear serum. The heart's muscle was soft and pale, and in places showed evidence of fatty degeneration. Small haemorrhages were seen immediately beneath the endocardium. The orifices and valves were normal.

The lungs were slightly emphysematous, but otherwise healthy.

The kidneys (8 ozs.) had their capsules closely adherent, and these, when stripped off, left a granular surface, evidently due to early interstitial nephritis. Microscopic examination

showed the appearance of a well-marked parenchymatous nephritis, as well as the interstitial change.

The liver, pancreas, and spleen were healthy.

The uterus was slightly larger than normal, and its mucous membrane was greatly congested.

The stomach was distended, and its mucous membrane soft and thin, due in part to atrophy and in part to digestion. The small intestine seemed healthy throughout. Microscopic examination of the stomach showed great atrophy and disintegration of the mucous membrane, there being few healthy epithelial cells made out.

The brain, spinal cord, and their membranes, to the naked eye, appeared quite healthy, unless for some slight congestion of the vessels. The pons, medulla, and cord were examined microscopically. They were fixed in formol (10 per cent. solution), transferred to alcohol, and cut in celloidin. Sections were stained (1) with theonin, (2) with haematoxylin and eosine, and (3) with osmic acid (1 per cent. solution). A careful examination was made of numerous sections taken from various levels of the cord, medulla, and the pons, and the only points in which these sections differed from the normal were (1) that the ganglion cells throughout the pons, medulla, and cord, contained a large amount of yellow pigment, and (2) that the majority of the vessels showed well-marked hyaline degeneration.

All degrees of pigmentation were seen in these ganglion cells, ranging from cells with only a few yellow granules to those whose plasma was entirely filled with the pigment (see plate). There were very few cells without some pigment, and at least 50 per cent. had as much as half their plasma filled with the pigment. In a small proportion of cells the yellow pigment was scattered throughout the cell, giving, under the low power, the appearance of a yellow ground-substance containing chromatic granules (Nissl bodies). In these cells the Nissl bodies were often paler than normal, and the cell altogether presented somewhat the appearance of a "ghost cell" with a yellowish colouration of its plasma (cells *j*, *k*, *l*, *m*). But by far the greater number of cells

had the pigment granules collected together into one part of the plasma, the rest being filled with well-stained Nissl bodies (cells *b*, *c*, *d*, *e*, *u*). And it is to be noted that in these cells the presence of large masses of pigment did not seem to affect the staining of the Nissl bodies, for, even when only a few of these bodies remained, their staining was quite intense (cells *o*, *p*, *r*, *s*). In the majority of the cells the pigment granules seem first to be deposited as a compact little mass at one pole of the cell. This pigment mass seems gradually to increase in size, displacing, as it does so, the adjacent Nissl bodies till, ultimately, the whole cell plasma is filled with pigment and no Nissl bodies remain. At the part of the cell where pigment and Nissl bodies join there was a certain overlapping of the two elements, and there the Nissl bodies seemed smaller in size, as if the invading pigment was breaking them up (cells *b*, *d*, *i*). With a lower power ($\frac{1}{8}$ th inch) the dividing line between pigment and Nissl bodies seemed sharply defined, but with the higher power ($\frac{1}{12}$ th inch) the above-mentioned fragmentation of the chromatophile elements could usually, though not always, be made out. It could not be said that the Nissl bodies at this line of contact were individually more deeply stained than at other parts of the cell, and so I did not recognize in these cells the "sclerozed zone" as described by Bevan Lewis. Neither could I find any cells corresponding to Bevan Lewis's first stage of pigmentation—cells slightly swollen, and staining deeply. Certainly some cells stained fairly deeply but they did not seem swollen, indeed they seemed quite normal.

It was difficult to determine with any certainty the effect of the pigment on the cell nucleus. But as far as I could make out the nucleus suffered in much the same way as did the Nissl bodies. That is to say, the nucleus seemed unaffected till actually invaded by the pigment granules, or, till the whole cell plasma was replaced by pigment, when the nucleus could sometimes be seen faintly outlined towards the centre of the cell (cells *j*, *n*). Probably, however, the nucleus was sometimes obscured by the surrounding

pigment, for in some cells where there was no appearance whatever of a nucleus, a few well-stained chromatophile elements were still to be seen in the cell plasma (cells *o*, *p*, *r*, *s*).

Very few cells were found presenting what might be called a primary chromatolysis, for the disappearance of the Nissl bodies in the vast majority of cells seemed to be due to the invading deposit of pigment and to no other cause.

As to the nature of this yellow pigment, little seems to be known. Under a high power it is seen to consist of numerous small granules, closely packed together in the cell plasma. These granules do not blacken with osmic acid, though they stain deeper than does the plasma of the cell, and for this reason, osmic acid staining is a very excellent method of demonstrating the individuality of the granules (cells *v*, *w*, *x*, *y*). Neither do the granules dissolve with ether, showing again that they do not represent a fatty degeneration.

As to the condition of the vessels in these sections, little requires to be said other than that throughout the whole pons, medulla, and cord they presented a well-marked hyaline degeneration. The change was most marked in the arteriols, but it was also seen in many of the larger arteries. All three coats of the vessels were found affected, but the degeneration was usually most obvious in the media, where often no appearance of muscle fibres was to be seen. The condition corresponds to atheromatous change found in larger vessels. No haemorrhages were noted, and in none of the vessels could it be said that their lumen had become obliterated.

These, then, are the morbid appearances found on examining the tissues of this case of tetany, and it now remains for us to consider what is their interpretation. The changes in the vessels indicate a degeneration, due most likely to the presence in the circulation of some toxic substance, the nature of which is unknown to us. Can it be that the pigment in the nerve cells has a similar explanation, and that this pigmentation of the cell is

a degenerative change? Such a view has been advanced. But another explanation, namely, that the pigment is the result or evidence of functional activity in the cell, is the one which seems to have obtained the more support. Bevan Lewis tells us that a deposit of yellow pigment is a constant appearance in healthy ganglion cells, and that it indicates a state of physiological activity rather than a pathological degeneration. He also points out that in the ganglion cells from cases of senile atrophy there is diminution of this pigment, while in cases of mania and epileptic insanity, where there was great motor excitement, the pigment is on the other hand excessive. He considers, therefore, that the pigment in ganglion cells is a sign of by-gone functional activity. If this, then, be so, the excessive amount of pigment in the ganglion cells of this case simply means that there has been a hyperactivity in their corresponding muscle fibres; and of this hyperactivity of the muscles the above report gives ample illustration. According to this view, then, tetany must be due to some poison which has a specially stimulating action on motor nerve cells, the activity of these ganglion cells being represented by an increase in their yellow pigment, and their hyperactivity by an excessive amount of this pigment as in the cells described above. But if this yellow pigment in the cell is evidence of by-gone activity, it must also mean a present incapacity, for we have seen that the pigmentation in many of the cells was so extreme as to entirely displace the Nissl bodies. Now, much importance is attached to the presence of these Nissl bodies, for they are thought to represent the functional potentiality of the cell, either in the form of nutrition, or as energy, or as both these combined. Whichever it may be, it seems sufficiently clear that the loss of the Nissl bodies must affect the function of the cell, be it that of a trophic centre or of a motor centre. And so it may be that, after all, this excess of pigment does represent the fatal lesion.

This extreme pigmentation of the ganglion cells is to all intents and purposes just another form of chromatolysis, by means of which the cell becomes a pigmented cell in place of becoming a "ghost cell." And careful examination

of large numbers of cells by means of high powers points to the pigment granules being formed from, or deposited as a result of, the fragmentation of the Nissl bodies. If there be any truth in this view—and I only suggest it, having no proof of it—the difference between the pathological anatomy of tetanus and tetany would lie in the different degeneration in the ganglion cell. In the one we have a chromatolysis producing a “ghost cell,” in the other another form of chromatolysis producing this pigment cell. And so if pigment in the cell does represent hyperactivity of the cell it also represents its atony.

As to where this poison in cases of tetany may come from, and as to how it gets at the ganglion cells, I have nothing to add to what will be found in the paper by Dr. Trevelyan already referred to. I have, however, greatly to regret that, for completeness, I did not examine the cells of the cerebral and cerebellar cortices. The necessity of so doing now appears much greater than at the time of the post-mortem examination, although I do not anticipate that any changes found in these cells could have greatly changed one's views as to the pathology of tetany.

In conclusion, I have to acknowledge my indebtedness to Dr. Steven, by whose permission, and at whose instigation, I publish this case. I am also indebted to Dr. Workman for access to the post-mortem room journal. I myself, however, am responsible for the account I give of the various microscopic examinations.

DESCRIPTION OF PLATE.

Cell *a*.—Normal ganglion cell.

Cells *b*, *c*, *d*, *e*, *u*.—Normal cells, except for deposit of yellow pigment. The pigment is confined to one pole of the cell, and the Nissl bodies are deeply stained.

Cells *f*, *g*, *h*, *i*.—Pigment diffused throughout the cell, the Nissl bodies not so well stained, and in places fragmented.

Cells *j*, *k*, *l*, *m*.—Pigmented “ghost-cells.”

Cells *o*, *p*, *q*, *r*, *s*, *t*.—Cells with plasma almost entirely filled with pigment; cells atrophied.

Cells *v*, *w*, *x*, *y*.—Stained with osmic acid to show arrangement of pigment granules.



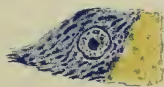
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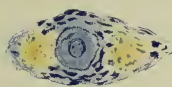
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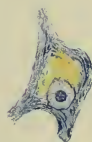
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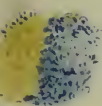
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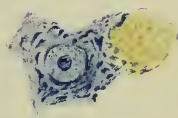
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v



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ON HYPNOTISM AND PSYCHIC IMPRESSION IN TREATMENT.

By ALEX. ROBERTSON, M.D., F.F.P.S.G.,

Physician to the Royal Infirmary, Glasgow.

PROBABLY in no department of medical science has progress been greater during the last thirty years than in the physiology and pathology of the brain and other nervous structures. Before that time a few leading facts regarding the functions of the cranial nerves, nerve-roots in the spinal cord, reflex and vaso-motor action, with very general conceptions of the relations of the mind to the convolutions, and of motion and sensation to certain regions of the brain and cord, summed up the most of what was known regarding the distinctive characteristic of the animal kingdom—the nervous system. Now, besides a large increase in our knowledge of the minute anatomy of this great system, we know much of the organization and functions both of its elementary and leading parts, so that in many cases, guided by the indications which disorder or destruction of these functions point to, we can localize with great accuracy the exact seat of lesion when one is present.

But while regarding with satisfaction the advances which have been made in our knowledge of nerve-centres and nerves in recent times, there is perhaps some risk of over-estimating its extent. After all, the known in this region, as in so many others, is not for a moment to be compared with what is unknown. It is when we come to consider the nature of nerve-force, the relations of organ and function, and, above all, the mysterious *nexus* of mind and brain, that the poverty of our knowledge and the depth of our ignorance become apparent. If this be true regarding mind and its cerebral

associations in their normal condition, as it undoubtedly is, even more so, if that be possible, does it hold good of abnormal manifestations, whether those of insanity or such states as somnambulism and hypnotism. It is with the last of these, hypnotism, that we are at present concerned. Although our subject leads us to consider it in its practical applications, a few preliminary remarks on the views that have been advanced regarding its nature, with a brief sketch of its history, may not be regarded as out of place.

Hypnotism was first brought under public notice by Mesmer, a Vienna doctor, who went to Paris in 1778, and there began to use it in the treatment of disease. So marvellous were the results that, it is stated, the French Government offered him £20,000 for his secret—for he held and used it as a secret remedy. This he refused to accept, preferring the *éclat* of the mystery which surrounded his remedy, and the golden harvest which it brought him. He alleged that connected with his own body there was an environment of healing magnetic fluid, which he had the power of imparting to others. However, two commissions which were appointed to inquire into his allegations and procedure pronounced against them, and after some years he left Paris, having previously fallen into general discredit.

It is interesting to find that Dugald Stewart in 1827, in his treatise on the "Philosophy of the Human Mind," fully recognizes the genuineness of the hypnotic phenomena. But it was not till 1842 that a trustworthy attempt was made to observe and record the facts with strict accuracy. Then it was that Braid, a Manchester surgeon, having had his attention drawn to the subject in his endeavour to expose what he at first deemed the jugglery of a travelling mesmerist, found that there was much of reality in the condition to which he afterwards gave the name of hypnotism. He then proceeded to investigate the question carefully, and, as the result of his observations and studies, came to the conclusion that there was a definite physiological change in the brain during hypnosis. He induced it by causing the subjects to gaze fixedly at something

bright between and above their eyes, so that a "double internal and upward squint" was produced. This, he held, had the effect of "paralysing the nervous centres in the eyes and their appendages, and destroying the equilibrium of the nervous system." And when the hypnotic state was developed, he attributed the special phenomena to the "power of imagination, sympathy, and habit in producing the expected effects on those previously impressed."

Braid's writings attracted much attention on the Continent, and ultimately the subject was taken up with enthusiasm in France, especially at Nancy, but also in Paris, at the Salpêtrière, by Charcot and others. Bernheim and his colleagues in Nancy, where hypnotism has been used in treatment on a very extensive scale, considered it to be a psychical state initiated by suggestion only, but probably producing vaso-motor changes. In his later writings Braid anticipated this and other of the views held by the Nancy school, some of which, according to Dr. Milne Bramwell, of London, he demonstrated to be fallacious. Dr. Bramwell¹ himself, whose experience of hypnotism has been very large, has arrived at the important conclusion, which he says was also Braid's opinion, that "crime could not be successfully suggested in hypnosis." Indeed, Bramwell states that "he has noticed increased refinement in the hypnotic state."

It only remains that we should briefly notice the theory of Mr. W. H. Myers, the Honorary Secretary of the Society for Psychical Research. Having regard to the fact that physiology has hitherto failed to suggest an explanation of the hypnotic manifestations, and looking at the question from a purely psychological standpoint, Mr. Myers submits as a working hypothesis that there are two realms of consciousness which he names supraliminal and subliminal. The former is the everyday or normal consciousness of life; the latter is, so to speak, a lower stratum, which ordinarily is not in action. "In hypnotism," to quote his words, "we gain instead of losing control. Instead of losing control over the supraliminal stratum, we gain

¹ *British Medical Journal*, vol. xi., 1898, p. 672.

control over the hypnotic stratum." According to him, the lower stratum is the realm from which the productions of genius emerge into supraliminal consciousness. The suggestions of the hypnotic operator appeal to this lower sphere of mind, and appear to "stimulate many sanative and recuperative operations whose results rise presently into the perceptions of our waking life."

It will be observed that this theory supposes that something is added to the normal state of being in hypnosis. While there are facts that appear to support this view, and some of the results which are recorded in the sequel of this paper show at least a restoration of lost power, it is still difficult to understand how the subjugation of will, which is so prominent a feature in the hypnotic state, should not be regarded as a loss while it lasts.

Proceeding now to the therapeutic aspects of the subject, we pass on to the record of cases in which hypnotism was used as a remedial agent. The patients were all under the writer's treatment in the Royal Infirmary, and were seen by many medical men besides students.

CASE 1.

M. S., age 24, shopwoman; admitted 8th January, 1899.¹ Complex neurotic disorder; recovery under hypnotism.

Patient's father is nervous; otherwise family history is good. She has had many neurotic disorders. When six or seven years old had convulsive fits at considerable intervals, but has had none since. About the onset of menstruation, at 14, she was subject to non-convulsive seizures, in which she was more or less unconscious, and these troubled her for three years, occurring generally about the menstrual periods. Nearly seven years ago she had an illness similar to the present one, but much less severe, for which she was treated in the Western Infirmary; it lasted about six weeks. Her recovery was apparently complete, except the hearing of the right ear,

¹Shown after recovery at a meeting of the Pathological Society, 10th April, 1899.

which was not restored, and ever since she has been quite deaf in that ear.

Patient's present illness has lasted about a year, and is ascribed to strain and anxiety about her work. At its commencement, all her fingers, with one exception, suppurated at the points. Soon afterwards she lost the power of both legs and one arm, the sight of right eye, as well as the power of speech and swallowing. There was temporary recovery of swallowing and speech, but about six weeks before admission they were again entirely lost. During this period she was fed with the stomach pump by her medical attendant thrice daily. Before the setting in of the two latter symptoms she screamed loudly for a short time. Menstruation had been irregular, but was not in abeyance. For many years she has suffered from bronchitis during a part of the winter.

Patient was seen by me in consultation with the family medical attendant in her own home. He told me that besides using electricity, especially the Faradic current, with great perseverance, he had practically exhausted all ordinary medicinal treatment, but without benefit. In view of these facts, and of the limited means of her relatives, she was advised to come into the Royal Infirmary so that she might be subjected to hypnotism. She consented with some reluctance.

The report in the ward journal bears that on admission she was well coloured and not much emaciated. Both lower extremities were completely paralyzed; she could not even move a toe of either foot. She could raise the right arm as a whole from the bed by the shoulder muscles, but could not bend the elbow or lower joints. The forearm was semiflexed on the arm, and the fingers towards the palm in similar degree, their respective flexor muscles being spasmodically contracted. The right knee jerk was in abeyance, the left greatly diminished. Full power of the left side was retained down to the pelvis; she had learned to write with the left hand. All forms of general sensibility were entirely absent on right side from the crown of the head to the sole of foot; on the left side they were not present in the lower extremity. Sight, smell, and taste in all their forms were absent on the

right side, but present on the left, with the exception of the eye, in which the colour sense was defective, as well as ordinary vision. The electrical condition of the muscles was tested in my presence by Dr. George Macintyre, and found normal to both currents. The vocal cords were observed to move freely on deep respirations. The functions of other organs were normal.

No treatment beyond slight massage was adopted for about a week after admission, as it was thought advisable that she should feel thoroughly at home in her new surroundings before beginning hypnotism. On January 15th she had her first séance. She passed into the hypnotic sleep after about twenty minutes' use of the method introduced by Braid himself, chiefly looking steadily at a small glittering box held near and a little above the eyes when the head was resting on a pillow in bed. While hypnotism was being established on this and succeeding occasions, it was steadily suggested to her that on awaking her nervous system would be stronger, and that she would recover the use of all her lost powers. Generally one or two were specially named, such as, on the first occasion, those of swallowing and speech. She was aroused in half an hour, though on after séances she was allowed to remain in the hypnotic state for an hour, and even two hours. Her return to consciousness was quiet and normal, undisturbed by any emotional or other disorder. Her power of deglutition was at once tested, and it was gratifying to find that she was able to swallow a little water. In the course of the day she took two meals herself, and from that time onwards there was no further need for the stomach-tube, as she had no difficulty in taking her food.

Next morning (16th) she was able to speak a few words in a low tone, and in two or three days her voice was fully restored. This and other defects when once they had begun to pass away showed no disposition to recur; on the contrary, improvement usually progressed to complete recovery.

The next séance was on the 19th January; a little delay, longer than was intended, had occurred, and she herself

requested to be again hypnotized. When aroused, the sight of the right eye had returned, and colour vision was not only normal in it, but was also nearly so in the left one. On the 23rd January, immediately after the third séance patient was able to move the fingers of the right hand and the two larger toes of the left foot. Between this date and March 4th there were six séances, and after each there was additional improvement in the patient's condition, not always at the time, in some cases not till next day. After the last of these séances, sensation was still absent below the right knee, though restored to all other parts of the body. The sensitive and insensitive parts of the limb were separated from each other by an abrupt line of demarcation. Without further hypnotism the sensory function in its varied forms gradually returned to the remaining part of the leg during the next eight days, progressing slowly downwards to the tips of the toes. It is worthy of note that the muscular sense in both limbs returned before any of the other forms of sensibility.

On the 18th February it was noted that patient was going up and down stairs and making herself useful in the ward, though sensation was then still absent on the right side. On March 5th, about 8.30 p.m., she told the nurse that she had pain in the right ear, which was present next morning at the medical visit. On again testing the hearing of this ear with the watch it was found that she heard its tick when applied close to the aural cartilage, but bony conduction was still in abeyance.

In the beginning of March patient had a severe bronchitic seizure, and this seriously interfered with the treatment of her neurotic disorder till the end of the month. However, with the exception of the right ear, the hearing in which was still very imperfect, there remained no vestiges of the complicated disorder of her nervous system, and she considered herself to be well. While contemplating returning to her home, there was a recurrence of pain in the right ear; but concurrently with it I found that there was a further marked improvement in hearing, as the tick of the watch

was now heard at a distance of six inches. She left the hospital on April 9th, but returned a week afterwards before going to the country to tell us that she continued quite well.

It will be observed that patient's present illness began with suppuration of all her fingers in succession, and this suggests that the disorder of the nervous system commenced in the trophic area. It is interesting to note how long a part of this system—namely, the auditory nerve and centres—may be out of function, and yet have its nutrition well maintained. She had been six and a half years absolutely deaf in the right ear, and now her hearing is all but fully restored. The fact of the recovery being associated with pain in the right ear is worthy of consideration.¹

CASE 2.

Agnes M., age 27, farm-servant, admitted into the Infirmary 5th February, 1896; hemianaesthesia and analgesia; failure of electricity; recovery under hypnotism.

Patient's family history was good, showing no disposition to constitutional disorder of any kind. She complained of pain in right side of abdomen in the ovarian region, and stated that it began eight years since as a "beating," which she could both feel and see. Though after two years it became less troublesome, it has never altogether left her since its commencement. About the menstrual period, and especially if the menstrual flux were scanty, this painful pulsatile feeling was worse, and extended over the lower abdomen and through to the back. It was frequently paroxysmal, occurring about three times a day and lasting for about three minutes on each occasion. She had never derived any benefit from treatment.

Patient had a healthy expression, and was in good condition.

¹ While this paper is passing through the press I learn that patient has had a relapse of some of her symptoms. She had remained well upwards of five months. Her relapse is attributed to strain at her work.

Pulse and respiration were normal; temperature was 99° in axilla; urine contained phosphates. There was no abdominal swelling; pain was complained of in the region of the right ovary, and this was increased by pressure. On the 12th February the report in journal bears that I noticed sensory defect on the entire right side, namely, complete analgesia with partial anaesthesia; folds of skin in the leg were transfixed with a needle without inducing pain or bleeding. The temperature and muscular senses were also in complete abeyance. Tested with the dynamometer the grasp of right hand registered 20 lbs.; the left 30 lbs.; patient was right-handed. There was thus some impairment of muscular power in right hand; though patient was not previously aware of it. With Faradic current there was vigorous muscular contraction on both sides; this was painful with even a moderate current on the left side, whereas a strong current was not felt on the right side. Taste on right side of tongue was absent; so was smell in right nostril; ammonia produced no impression on this side of the nose, and the other mucous surfaces on the affected side were anaesthetic. Dr. Napier oculist, reported and showed on diagrams that the visual field was contracted very much in right eye and slightly in left one, and that there was great defect of colour vision on the right side. Hearing distance as tested by watch was 1 inch on right side, normal on left.

On February 22nd it was noticed that there were several sensitive patches of integument on the right side—one 2 inches in length and $\frac{1}{2}$ inch in breadth on the face, extending from the lobe of the ear along the line of the jaw; one beneath the right clavicle; one beneath inner canthus of eye; and one, 1 by $1\frac{1}{2}$ inches, near costal margin, about 3 inches from middle line. A small sensitive area was also found on the leg.

Medicinal treatment with electricity, both Faradic and continuous currents, was used for about a fortnight, but without benefit. Being wishful to try the effect of a powerful electro-magnet held close to the anaesthetic skin without actual contact, on February 24th I had patient removed to the

electrical room of the hospital, and there moved a powerful horse-shoe magnet—powerful enough to suspend an ordinary poker and tongs together—along the affected limbs and side of the body, occasionally, though not intentionally, touching the skin.¹ There was no improvement in sensory defect. On the same day a strong Faradic current was freely applied to the anaesthetic arm by the wire-brush without effect, more than that she had a feeling of cold where the application was being made, which immediately passed away. No result followed the application of gold, silver, and bronze coins to the affected parts. The patient's temperature, taken in the axilla, before these observations were made was 97·4, and at their close 99·2.

On 9th March the electro-magnet was again used. It was first applied to the right arm, but for some seconds there was no effect. The arm was then touched with the magnet for two or three times momentarily. Prior to the application it was suggested strongly to her that feeling would return. Accordingly, after the magnet touched her, tactile sensation was restored. But, on testing, it was now found that there was considerable but not complete anaesthesia of the *left* arm. At this time there was no change in right face, thigh, or leg; but on bringing the magnet very near these parts consecutively, *without touching them*, tactile feeling after the lapse of about twenty seconds returned to them all. There was also restoration to painful impressions, but not so completely. Her ability to distinguish colours was improved, but she still mistook red for dark-blue and *vice versa*. She also heard better with right ear, though hearing with this ear was still imperfect.

No further observations were made for three days—till 12th March. The tactile function was then found to be deficient on both sides, the left being worse than the right. Moreover, the thermal sense was equally defective, a very hot test-tube being felt as cold on the right as well as on the left side. Taste on both sides of the tongue was normal; but sense of smell, at all events to assafoetida and pepperment,

¹I was indebted to Professor M'Kendrick for the use of this magnet.

was in abeyance in both nostrils. She distinguished colours more correctly with the right eye than with the left. On the other hand the hearing distance was 14 inches in the right ear and two in the left (watch); this, it will be observed, was the reverse of what it was on admission, the left being then normal. There was no pain on pressure over the left ovary, but it was still marked over the right one.

On the 17th March sensibility in all forms, except heat, which was felt as cold, was present on right side of face, arm, side of body and leg, but not in sole of foot. There was, however, an area between the umbilicus and the pelvis in which tactile and painful sensibility were not restored. On the left side sensation was in abeyance, except thermal impressions, which were all felt as cold.

On the 18th March the electro-magnet was applied to left forearm and arm, but without result—sensation remained absent.

On March 19th patient was hypnotized, and allowed to remain in the hypnotic sleep for an hour. A little difficulty was found in arousing her, and when she did awake she was excited, struggled, and talked incoherently. She also vomited. Her mental disturbance did not last more than five minutes, when she became quite rational. It was not then thought advisable to test her sensory condition.

On 20th March sensation everywhere was found to be quite normal. This was clear in regard to touch, pain, heat, and cold, and the muscular sense. Her colour vision was all but perfect; the only mistake she made on being tested by coloured wools was naming grey yellow with the left eye. There was slight remaining pain on pressure over the right ovary.

On the 23rd March she left the Infirmary, being then apparently quite well. From time to time during the next six or eight months she wrote to the nurse of the ward telling of her progress. She had returned to her employment, and continued in good health, free from her neurotic disorder, except a short return of anaesthesia in one of her legs, which had not, however, prevented her from working.

CASE 3.

Primary dementia and motor paralysis; improvement under use of myelin and suggestion; complete recovery after hypnotism.

As this case has been already published at length,¹ only a summary of it will be recorded here. A. W., age 39, single, railway signal fitter. He was admitted into the Royal Infirmary on 2nd February, 1894. He was a strongly built, healthy looking man, but his medical history showed that he had neurotic illnesses previously of a similar kind, though less severe in degree. His habits were temperate, and there was no evidence of syphilitic disease. His family history was good. His present illness had only been of about eight days' standing before admission, and no cause for it was known. It had begun with headaches, giddiness, and weakness of the legs, so when admitted he could not walk without assistance. Memory and intelligence had failed greatly. He lay in bed with a dull, absent expression, and his speech was somewhat thick. The grasp of the dynamometer was 6 lbs. with each hand, whereas the average for a labouring man with the same instrument was 40 to 50 lbs. The superficial and deep reflexes were present in the lower extremities, the latter somewhat exaggerated. Sensation was not impaired. There was no weakness of bladder or bowels. Other functions were normal, except the appetite, which was poor.

Patient had a variety of ordinary treatment, but did not improve much till he was put on myelin² on March 3rd. Under its use there was return both of mental and physical power to such an extent that he was able to leave the Infirmary on 21st April, intending to resume his employment. He was not,

¹ *Glasgow Medical Journal*, October, 1895. Some months after recovery this patient was shown to the Eastern Medical Society. He was then in excellent health.

² Myelin was the fresh brain of the sheep mixed with aromatics, and was prescribed by the writer in various functional disorders of the nervous system (*British Medical Journal*, 26th December, 1893). It has since then been superseded by cerebrin.

however, considered to be well, as mentally he was depressed, and his legs were not strong.

On 27th October he was re-admitted. He stated that after his dismissal in April he was only able to work one week, on account of weakness in his legs. His mental condition, however, had not suffered seriously, though depression remained, and was still present in a marked degree. Under the use of the myelin, with suggestion of recovery, there was again improvement, but obviously he was not well in regard to either mental or physical power. On 5th December he was hypnotized without difficulty, but was aroused after ten minutes; suggestions of complete recovery both of mind and body were made to him before and during the hypnotic state. On the following day he had a brighter expression of countenance, and his mind was clearly more active. Three days afterwards he was again hypnotized. This was followed by complete recovery. He was kept under observation in the ward till December 21st, when, there being no disposition to a relapse, he was dismissed well.

CASE 4.

A. W., age 24, domestic servant; admitted 20th March, 1898; motor, sensory, and mental phases of hysteria; recovery under hypnotism; partial relapse. This case has been summarized from the much-detailed report in the ward journal.

Patient's father is a habitual drunkard; she has been subject to much strain and worry. She is of a quiet, calm disposition, but very sensitive. About five years since became affected with hysterical convulsive seizures, and at first had them at intervals of about six weeks, but for eighteen months before admission, when she had one, there was entire immunity from them. When about twenty years old she became affected with ptosis of both eyes, one being worse than the other. On this account a portion of both upper eyelids had been removed, and since then has been unable to close one of her eyes completely. During the month of April had several

convulsive seizures, and in the intervals was troubled with winking movements of the eyes and tremulous action of the labial muscles. There was brief delirium after some of the fits, during which she fancied she saw a man, apparently always the same one, seeking to do her harm. There was also temporary defect of feeling in the arms. Hypnotism was begun on 12th April, and continued afterwards at intervals of about a week till 22nd May, when it was noted that as a rule she did not feel better at the close of the séance, but that next day or the day following the twitchings of the face and sensory defects were much less. She herself thinks that hypnotism does her good, and asks to have it repeated. On June 25th she had her last séance, and at its close she drew attention to the fact that twitchings of the mouth and eyes had ceased. On the following day she left the hospital apparently well, and soon afterwards returned to domestic service.

On the 11th August patient was readmitted, her nervous twitchings having returned. It appears that she had been subjected to excessive work as a servant. Convulsive seizures occurred occasionally, and areas of hyperalgesia and anaesthesia were irregularly distributed over body and limbs. Colour sense was largely in abeyance. She recognized the taste of salt, but not of sugar, on both sides of tongue. Thermal and electric anaesthesia were present in forearms, but muscles contracted well to Faradic current. There was marked contraction of visual fields. She was twice hypnotized, on 24th and 25th September respectively, and was apparently improved for a few days. But it was on the 5th October that the more marked sensory defects were observed, showing that hypnotism had a very brief controlling influence, if any, on the neurotic phenomena during this illness. After this, patient was left without special treatment beyond tonic measures. She slowly improved, and ultimately was dismissed on January 5th, 1899, with slight remaining sensory defects in her arms. Since then she entered into the service of one of the officials of the Infirmary, and up to the present time (August) has continued at work. I know that she is regarded by her employer as a most

faithful and trustworthy maid. She has, however, had a relapse of the ptosis to some extent, and at that time I saw her and reassured her of its passing away, which it did after a few days. I learn that she had also anaesthesia of one foot, which also disappeared in two or three days, and did not prevent her from attending to her work.

PSYCHIC IMPRESSION.

This is the title I have given to methodical or systematized suggestion in the treatment of functional disorders of the nervous system. As far back as 1871 I tried the effect of strong mental impression in insanity, and recorded my experience in the *British Medical Journal* for that year. It was not encouraging, but this is attributable to its having been used in confirmed mental disease, over which I fear it has no influence. Its proper sphere of usefulness is in the slighter forms of derangement of the nervous system, particularly those commonly classed as hysterical, but which are better named neurotic.

The method of procedure is as follows:—Sitting down opposite the patient if in bed, or standing opposite her—for in most but by no means all cases it is a woman—if on foot, I require her to look steadily at my eyes. This frequently causes a difficulty at first, for there is a strong disposition for the patient in such cases to look down or aside, but on speaking resolutely she eventually complies with my request. Then, continuing in a firm tone of voice and decided manner, I address her somewhat in the following terms:—You understand that these seizures—if subject to spasmodic or other form of attack—must stop. This I repeat, perhaps modifying the remark, till I get the patient to say, “Yes, Sir.” Daily or every second day at gradually increasing intervals this formula is gone over, and even after the patients have left the Infirmary I have required them to return once a week for several weeks, so as to maintain the controlling and fortifying influence on the mind and brain.

For several years this mode of treatment has been carried

out in the wards of the Infirmary under my charge, in a considerable number of patients, male as well as female, but chiefly the latter. The results in most cases have been immediately successful, and have been observed by medical practitioners as well as students. It is not, however, sufficiently powerful in its action to cope with the more severe forms of neurotic disorder, such as those recorded in the early part of this paper. Nor is it so strong an agent as would lead us to expect much result from it in the treatment of most cases of insanity. Still in some of the slighter forms it may prove of use. In one case of chronic melancholia in which no benefit was derived from the administration of thyroid extract and cerebrinin, the daily practice of psychic impressions by myself in the way described, so far as I could judge, clearly assisted ordinary general treatment in inducing recovery.

The results of hypnotism in the first three cases recorded in this article were very satisfactory, and in the first two particularly gave striking testimony to its value as a therapeutic agent. In Case 2 the failure of electricity in its most powerful forms to do more than induce a transference of the sensory defect from the one side to the other, with the immediate success of hypnotism in the restoration of normal function was very impressive. In Case 4, on the other hand, mere temporary benefit followed its use, and it may well be doubted if the patient's present fairly satisfactory though somewhat unstable condition has not rather been the result of ordinary general measures than of any of the special agents employed.

It may be right that I should state my general impression that no prejudicial effect followed the use of hypnotism. Care was taken in all cases to warn the patients against permitting themselves to be made the subjects of simple experiment. Even for medical reasons it should only be had recourse to when ordinary medical measures have failed. In closing, I again direct attention to the value of systematized psychic impression in the slighter forms of functional disturbance of the central nervous system.

ON THE CLINICAL SIGNIFICANCE OF DOUBLE OPTIC NEURITIS IN CHILDREN AND YOUNG ADULTS, AND ON THE CAPACITY FOR RECOVERY FROM SYMPTOMS OF TUMOUR OF THE BRAIN.

By C. O. HAWTHORNE, M.D.

THE cases which form the basis of this paper illustrate some of the circumstances under which double optic neuritis affecting children and young adults occurs in clinical practice. In none of them, fortunately, is the cause of the neuritis made absolutely certain by post-mortem examination, but as they are records of clinical facts which demanded to be dealt with both from a diagnostic and prognostic point of view, they have a definite practical interest and value. An attempt has been made to increase this value by making the records as complete as possible, both by detailed examination of the patients, and by following the cases after the opportunity for continuous observation had passed away. In this respect it may be noted, that whilst some of the cases were first seen when the symptoms, including the optic neuritis, were of recent development, others came under observation long after—in some cases years after—the acute conditions had subsided. Hence, presuming these two groups include cases of the same order, the clinical picture they contribute to form is rendered more complete, the probable issue of the earlier cases being reasonably inferred from the known facts of those seen at a later stage. At all events, the records convey some of the considerations and difficulties which arise in the practical handling of such cases, and at the same time contribute some-

thing to our knowledge of the clinical history of optic neuritis.

The patients are all young, their ages at the date of the first observation ranging from 10 to 21 years, but the date of the occurrence of the optic neuritis was not in any case later than the seventeenth year. Of the total of nine, six are females and three males.

The first question which inevitably arises on the recognition of a double optic neuritis is this—Is the neuritis due to a tumour of the brain? the word tumour being used, in the sense Hughlings Jackson defined it, as equivalent to a “foreign body” or “adventitious product” within the skull. An affirmative answer to this question is sometimes easy; at other times such a result can only be reached if all the evidence is carefully collected and weighed. On the other hand, a confident negative answer is almost always difficult, and for the most part is only justified when some other admitted cause for the optic neuritis can be certainly recognized, or when, in the absence of any such cause, and in consequence of the lapse of time, the progress of the case, and the non-appearance of other evidence, the existence of a tumour is rendered highly improbable. The diagnostic position thus defined is the first practical issue from a study of the cases here under consideration.

Attention may now be directed to the evidence of cerebral disturbance actually present in the first seven cases, which is taken as justifying the view that the optic neuritis was in all probability dependent in each case on the existence of an intracranial tumour.

These seven cases naturally fall into two groups—(1) those seen shortly after the development of the optic neuritis; (2) those in which the optic neuritis had long since passed into a condition of optic atrophy.

The first case (see p. 130) presents evidences of cerebral disturbance in abundance. Obstinate vomiting, and pain in the head varying much in intensity, are sufficient of themselves, together with the optic neuritis, to make the diagnosis, but

here we have in addition, giddiness, retraction of the head, anosmia, and certain paralyses, not to speak of the mental phenomena and the slow pulse and subnormal temperature.

The second case offers a marked contrast to the abundant evidence provided by the first. With the exception of considerable pain in the head and optic neuritis there is no manifest evidence of cerebral disturbance, yet these will be allowed to be sufficient, and more especially as they receive confirmation from a study of the knee-jerk. The observation in this case was made under unusually fortunate circumstances, for whilst the knee-jerks were present when the boy was first seen, they disappeared during his residence in hospital, and they have never since been detected. There is thus no doubt that the patient lost his knee-jerks during the time when he was the subject of headache and double optic neuritis almost certainly due to cerebral tumour. In two other of the cases (Nos. 5 and 7) here submitted with a diagnosis of intracranial tumour the knee-jerks were absent. No doubt before such a conclusion in reference to the knee-jerk is justified the examination demands great care, as unless this is exercised the jerk, though potentially present, may not be elicited. In examining these cases the precautions insisted on by Jendrassik, Buzzard, and Gowers have been borne in mind, and whenever possible the tests have been repeated and confirmed by other observers. The result is to show that of the seven cases the knee-jerks were absent in two (Cases 5 and 7), in one (Case 2) they disappeared under observation, and in another (Case 1) they varied at different stages of the case, being sometimes present or exaggerated and sometimes absent or diminished.

The point of importance just now is the restriction in Case 2 of the evidences of cerebral disturbance to double optic neuritis, headache, and loss of knee-jerks, and the necessary inference arising therefrom, viz., that such loss may be a valuable confirmatory sign of intracranial disturbance. Yet it must be remembered that the loss of the knee-jerk did not occur until fully two months after the onset of symptoms; during the greater part of these two months the boy had no

complaint to make except of pain in the head, and it was not considered necessary to seek medical advice until sight was failing. The earlier phases of the case were marked by nothing more than headache and optic neuritis. It is highly probable, therefore, that neglect to use the ophthalmoscope at that date would have led to a serious error of diagnosis.

In Case 3 a further illustration is afforded of the slightness of the evidence, apart from optic neuritis, in some cases of cerebral disease, presumably tumour. Though the boy had never been confined to bed even for a single day, yet symptoms of intracranial disturbance had undoubtedly existed for six or seven months. Every morning during that period the boy vomited and had some frontal pain, but at other times he appeared quite well. When medical aid is sought the boy is blind from optic atrophy, the result of neuritis which had doubtless existed for months. Here again the first, and for long the only symptom, was vomiting, and that limited to one particular hour of the day. The vomiting and double optic neuritis were the only two events which occurred in a period of ten months, the vomiting being regarded by the parents as a trifling affair and only mentioned under some pressure. Yet it was the first, and probably for some time the sole evidence of an organic intracranial disease which has resulted in the permanent destruction of the patient's sight.

The three cases well display the cerebral events which may be associated with double optic neuritis as evidence of (presumably) intracranial tumour, and show how widely such events may differ both in their number and severity.

The next four cases came under observation for defective sight dependent on optic nerve atrophy, the conditions having been in existence for periods of time ranging from six months to ten years. In all but the last one the optic atrophy was manifestly the sequel to an optic neuritis, and the question may be raised whether this case (No. 7) ought to stand in line with the others. And all the more so, that whilst in the three other cases (Nos. 4, 5, and 6) there is a distinct history of a more or less severe illness with such symptoms as pain in the head and vomiting, the case now alluded to has no such history,

but, on the contrary, the failure of sight is reported to have come on quite gradually and without any other symptom or evidence of any kind. There are, however, certain reasons which may be urged in support of the suggested diagnosis. In the first place, the absence of evidence of neuritis at the present date does not prove that neuritis never existed. "I am certain," writes Hughlings Jackson,¹ "that the most striking appearances of double optic neuritis . . . will pass away so far as to render it impossible for a careful observer seeing the patient for the first time, when recovered, to be able to declare that the peculiarities discoverable about the optic disc are pathological." Similarly, Gowers² writes, "Great caution is necessary in inferring, from the appearance of discs long after the onset of the atrophy, that this was simple and not neuritic; . . . the characters of the latter may ultimately resemble very closely those of the former." The same point is emphasized by Buzzard.³

There is another statement resting on the authority of Hughlings Jackson⁴ which may be advanced in support of an arrangement that places Case 7 in line with the previous six. It is this, "There is sometimes an acute illness as our first evidence of tumour . . . but it is important to bear in mind that optic neuritis may occur without any headache and without vomiting, or that headache may be slight and the vomiting none." It must therefore be regarded as possible, that the condition of the optic discs in this patient is not due to a primary atrophy, but is subsequent to a pre-existent optic neuritis which was unaccompanied by any other sign of cerebral disturbance, and that the evidences of neuritis other than the atrophy of the discs have disappeared. But even though the atrophy of the optic nerve were primary in character this would not exclude the diagnosis of cerebral tumour, though it is difficult to think of such a result except from a tumour situated so as to press on the optic

¹ *Medical Times and Gazette*, 1873, ii., p. 541.

² *Medical Ophthalmoscopy*, 3rd edition, p. 166.

³ *Diseases of the Nervous System*, p. 148.

⁴ *Transactions of the Ophthalmological Society*, 1880-81, vol. i., p. 72.

chiasma,¹ whereas in the case now under discussion, the presumption from the evidence is that the tumour is in the cerebellum. The point, however, need not be further laboured, as the evidence of intracranial disturbance is by no means limited to the condition of the optic discs. Further and sufficient proof is provided by the existence of nystagmus, by the absence of the knee-jerks, by the occurrence of "fits" of the *petit mal* order, and by outbreaks of temper which may be taken as evidence of depreciation of the cerebral tissues. Attacks of a similar character are noted in Case 6, where the diagnosis of tumour can scarcely be questioned (see also Case 2).

Taken together, the four cases of the second group confirm and extend the lesson enforced by the members of the first group. They show that the degrees of cerebral disturbance which accompany optic neuritis of intracranial origin may vary widely, and they further demonstrate how largely the results of such disturbance may pass away, though the injury to the organ of sight may be both permanent and pronounced.

It is possible that the question may be raised whether after all these cases are really cases of intracranial tumour, and especially in view of the evidences of recovery which they exhibit. And it may be admitted that reasoning on *à priori* grounds, or considering only the numerous undoubted cases of tumour in which the patients suffer severely, and which pass with comparative rapidity to a fatal termination, there is some difficulty in accepting the diagnosis here suggested. But it cannot be denied that in all the cases there is evidence of definite organic mischief within the skull, and the facts of experience do not seem to offer any choice other than tumour or meningitis. Taking the mere occurrence of recovery into consideration, it is certain that the diagnosis of meningitis, at least of tubercular meningitis, is not less difficult than that of tumour. Then in three of the cases the patients never had in the general sense of the term any illness which could reasonably be regarded as an attack of meningitis, and in those who were at any time seriously ill there were in two cases signs of

¹ Handford, *Brain*, 1892, vol. xv., p. 463.

Hinshelwood, *Glasgow Hospital Reports*, vol. i., p. 4.

a localized lesion in the brain, such as paralysis of a limb or a hemiplegic attack. Possibly, though certainly very rarely, such paralyses may occur in meningitis, but the presumption from such incidents is decidedly in favour of tumour. Again, it is to be remembered that in several of the patients the evidence of active intracranial disease extended over many weeks, and even months—a state of matters difficult to reconcile with a diagnosis of meningitis. And if the cases were cases of tubercular, and presumably therefore of basal, meningitis, the absence of disturbance of the functions of the ocular muscles is certainly remarkable. Further, in the one patient seen throughout the whole course of her very serious illness the temperature, so far as it was disturbed, had a range below the normal level, and two other members of the series seen with more or less acute symptoms were also free from evidence of fever. The degree of the optic neuritis is also of importance in the discussion of this question. Optic neuritis is undoubtedly present in a certain number of cases of meningitis, but it is almost invariably of slight, or, at the outside, of moderate development. But in all the present seven cases—with the possible exception of the last—the degree of optic neuritis either was, or had been, of the considerable or extreme degree usually found with, and indeed strongly suggestive of, intracranial tumour. The balance of probability, even when attention is confined to the facts of the present cases, thus strongly inclines to the diagnosis of tumour rather than to that of meningitis. Other considerations support this view. Thus it is a fact that a tumour, or even tumours, may develop in the brain and yet fail to give the slightest clinical evidence of their existence. Such growths, too, may be of considerable size, and may occupy parts of the brain which, to judge from general experience, are by no means tolerant of disturbance. The cerebellum, the frontal lobe, the occipital lobe, the temporo-sphenoidal lobe, even the motor tract, has each in turn been found to be the seat of a tumour which during life gave no evidence of its presence.¹ A step in advance of these cases is provided by a further series in which, with a tumour

¹See Table I., p. 126.

of the brain (proved by post-mortem examination), there has existed some, but only slight, evidence of cerebral disturbance, the evidence being in some of these cases equivocal or non-suggestive of tumour.¹ A further contribution to the argument is found in records of cases in which severe symptoms of brain tumour have subsided, to be followed after an interval, and sometimes a long interval, by return of the symptoms and death of the patient, the diagnosis of tumour being confirmed by post-mortem examination.² Indeed, the recorded instances of verified cases of brain tumour form a series in which, commencing from those absolutely without symptoms, there may be traced all degrees of severity in the number and degree of the disturbances produced.

In reference to the question of recovery, Hughlings Jackson³ writes that "it is not at all uncommon for a patient to get rid of all symptoms of tumour except that he remains blind or defective in sight," and in discussing the diagnosis as between meningitis and tumour adds, "But when recovery follows, what are we to say? . . . I still think them to be cases of tumour." T. K. Monro⁴ has recorded a case which comes as near actual demonstration as possible of the recovery of a patient from the effects of a brain tumour with the exception of optic atrophy and blindness. The patient, a man of 63 years, died in the Glasgow Royal Infirmary from cancer of the stomach. He was the subject of optic atrophy, and was almost blind. For thirty-three years he had been an inmate of the Blind Asylum, the loss of sight being attributed to a severe illness which confined him to bed for almost twelve months when he was 16 years of age. The knee-jerks were normal. The post-mortem examination showed a myxomatous tumour almost replacing the left half of the cerebellum, with atrophy of the optic chiasma and nerves. Such a record materially strengthens the decision for a diagnosis of tumour in such cases as are now under consideration.

Thus the facts of the present cases, together with the pre-

¹ See Table I.

² See Table II.

³ *Medical Times and Gazette*, 1873, ii., p. 197.

⁴ *Glasgow Medical Journal*, 1896, ii., p. 173.

sumption arising from a study of other cases, form a strong foundation on which the diagnosis of intracranial tumour seems securely placed. It would certainly be difficult to find anything like so strong a defence for a diagnosis of meningitis.

In reference to the disappearance of symptoms that have become fully established, it has to be borne in mind that the usual clinical effects of an intracranial tumour are due not so much to the pressure or mechanical displacement which the tumour produces as to the vascular and inflammatory disturbances which are excited in its neighbourhood. Given cessation of growth of the tumour, it is to be expected that such disturbances will subside, with a corresponding diminution in the symptoms due to them.

Presuming then that these seven cases are examples of the clinical exhibition of the effects of intracranial tumour, two conclusions follow. First, as already observed, the physician must be prepared to expect in different cases very different amounts of evidence—sometimes of such a nature and degree as to proclaim loudly the existence of intracranial disease, at other times slight in amount, and not manifestly cerebral in origin. It is in these latter cases especially that the ophthalmoscope renders such useful service, for whilst optic neuritis is not present in every case of brain tumour, it exists at some stage or other in the great majority, and not infrequently it is an early, if not the earliest, evidence. Even when existing alone, and more especially when it affects both eyes and is distinguished by much swelling of the papilla, it demands the consideration of cerebral tumour as a possible diagnosis, though, as will be shown later on, it may have exactly the characters just described and yet be due to some other and much less serious cause than tumour of the brain.

The second conclusion which follows from the acceptance of intracranial tumour as a diagnosis in the seven cases now under discussion is, that the prognosis of such cases so far as life is concerned is by no means hopeless. One patient has survived the loss of sight by eleven years and enjoys good general health except for an occasional quasi-epileptic attack, another

has a similar experience extending over four years, a third for three years, and the rest, though less impressive, are similar in the direction of their testimony.

Several parallel series to the seven cases now submitted are on record. So far back as 1866 Mr. Hutchinson¹ tabulated a series of twelve cases of optic neuritis in children following as a rule a more or less severe illness marked by delirium and other head symptoms, and he was able to show by the subsequent histories of the cases that frequently such children regain good health excepting as regards sight. Mr. Hutchinson² also reported a number of similar cases in 1879. The precise nature of the primary lesion he considered doubtful, but was disposed to distribute the cases among tubercular meningitis, basal arachnitis, cerebro-spinal meningitis, and some of the exanthemata, as the probable causal conditions. T. K. Monro³ has published three cases of blindness following double optic neuritis other than the one already quoted, and regards each case as one of intracranial tumour. Indeed it seems difficult to avoid that conclusion in reference both to his and other similar cases unless the accepted clinical significance of the combination of pain in the head, vomiting, and double optic neuritis is to be seriously challenged.

The possibility of recovery from symptoms, and even severe symptoms, of brain tumour must necessarily be considered in determining the line of treatment to be pursued. The recognition of such a possibility cannot fail to infuse not only a more hopeful tone into the prognosis but also increased resolution into the treatment. Probably most will agree that when the diagnosis of intracranial tumour is accompanied by localizing symptoms, surgical interference should be anxiously considered; that in any case where this is not deemed advisable medical treatment must consist of attempts to deal with the more troublesome symptoms; and that in the event of recovery occurring the measures indicated are those

¹ *Royal London Ophthalmic Hospital Reports*, 1866, vol. v., p. 307.

² *Ibid.*, 1879, vol. ix., p. 124.

³ *Glasgow Medical Journal*, 1897, ii., p. 367.

calculated to promote and secure the general vigour of the patient. Recovery however is a matter of degree, and at least two (1 and 5) of the present cases illustrate the dangers which may attend the patient even long after the original illness has subsided—a fact important to the prognosis, and also significant in reference to the anxious care with which these patients should be guarded. Hughlings Jackson¹ is especially emphatic upon the great necessity for caution in framing the prognosis in any case presenting symptoms of intracranial tumour. He points out, not only the liability to return of the symptoms after a period of quiescence, but also the very extended duration of the symptoms in occasional cases. Another danger on which he insists is that of sudden death, which may occur even when the patient appears to be doing well. The extent of this last risk receives striking illustration in a careful and elaborate analysis of 100 cases of brain tumour published by Hale White.² There is another point to consider in reference to treatment. All the patients referred to in this paper as examples of recovery from the symptoms of intracranial tumour have this very serious qualification of their recovery, namely, that each one is blind. Recognizing that this is a very likely issue, that it may indeed be a relatively good result of the case, the best perhaps to be hoped for, it is of great moment to consider whether any means can be adopted to secure the patient against so grave a defect. Hughlings Jackson has considerable confidence in large doses of potassium iodide with or without mercurial inunction. There seems no doubt at all of the fact that trephining the skull, altogether apart from the removal of a tumour, is sufficient, at least in some cases, to cause the subsidence of the optic neuritis. This measure has been advocated both by Victor Horsley and Macewen,³ and Jas. Taylor⁴ has also called attention to it. The question is one of great importance, for, from such cases as are recorded here, and from the statement that

¹ *Op. cit.*, p. 195 *et seq.*

² *Guy's Hospital Reports*, 1885, p. 117.

³ *British Medical Journal*, 1893, ii., p. 1365.

⁴ *Transactions of the Ophthalmological Society*, 1884, vol. xiv., p. 105.

the neuritis will subside under trephining, there would seem ground for hoping that in a certain number of cases of intracranial tumour where the situation of the growth cannot be localized, operative means may still assist in promoting the patient's comfort, and given a natural tendency for the growth of the tumour to cease, may assist him to many years of good general health and unimpaired vision. One difficulty in urging on the patient what he at least will consider heroic measures is that occasionally all the evidences of intracranial tumour, including optic neuritis, subside spontaneously.¹ Another arises from the fact that optic neuritis may occur apart from intracranial tumour, and whilst at the time of its development and existence it may be impossible to say tumour is not the cause—for undoubtedly optic neuritis may be for long the sole evidence of tumour—the subsequent history of the case renders such a diagnosis in the highest degree improbable.

This latter difficulty is illustrated by Cases 8 and 9 here recorded. In Case 8, not only was there double optic neuritis, but there was also paralysis of the right and possibly slight impairment of the function of the left sixth nerve, an event under the circumstances as significant of cerebral disturbance as well could be. Was such a combination indicative of tumour? In the early stage of the case it seemed impossible to deny that tumour might be the explanation, but the subsequent history renders such a diagnosis very improbable. There were no cerebral symptoms other than the optic neuritis and the ocular paralysis, but it was difficult to conceive of any explanation of these apart from intracranial mischief. No doubt the girl was anæmic, and anæmia is said to be one of the causes of optic neuritis. But anæmia, unless indirectly through the agency of a thrombosis or hæmorrhage, will scarcely produce a sudden diplopia and complete paralysis of a cranial nerve; and optic neuritis is itself a very rare and unusual circumstance in anæmic patients. An examination of every patient admitted to Sir Wm. Gairdner's wards for a period of five years did not reveal a single instance of the occurrence of optic neuritis with

¹ See Table II.

simple anæmia or chlorosis. Considering how very common cases of chlorosis are, how not only relatively but absolutely rare it is to find optic neuritis accompanying that condition, it becomes a question whether, when such association does occur, some new factor has not complicated the problem. The possible gravity of the significance of double optic neuritis certainly renders it most inadvisable to consider the mere existence of anæmia an adequate explanation of its presence, at least until all other known causes have been excluded, and even then, as already remarked, the extreme rarity of the association of the two conditions justifies some suspicion of the accuracy of their suggested relationship.

The case (No. 8) is an example of double optic neuritis existing with a circumstance strongly suggestive of intracranial mischief (namely, paralysis of the sixth nerve), yet almost certainly the case is not one of tumour. The difficulty of providing a satisfactory diagnosis is great. A very similar case is recorded by Gowers,¹ but the patient, in addition to optic neuritis and sixth nerve paralysis, suffered from attacks of severe occipital pain and vomiting, and the view taken of all these symptoms was that they were due to a transient localized meningitis. Possibly some help in understanding the present case (No. 8) is afforded by some observations recorded by Bristowe² noting the development of optic neuritis in anæmic patients, with evidence of thrombosis of some of the cerebral sinuses and veins. One patient, a girl of 19 years, when under treatment for anæmia, had a severe pain, first in the neighbourhood of the right ear, and later, a similar experience on the left side, with, on each side, tenderness and swelling suggesting thrombosis of the internal jugular vein. She was found to have double optic neuritis, and vomiting and headache were troublesome. After a few days she improved, but convalescence was interrupted by an attack of phlebitis in the leg. Bristowe's suggestion is that the attacks of pain about the ears meant thrombosis in the lateral sinuses, and he supports this by the record of a second case.

¹ *Transactions of the Ophthalmological Society*, 1880-81, vol. i., p. 115.

² *Clinical Lectures and Essays on Disease of the Nervous System*, p. 184.

This patient, a woman, æt. 23 years (six weeks pregnant), suffered for twenty-five days from headache, sickness, pain and tenderness over the left mastoid, and general drowsiness; during her illness she developed a right hemiplegia, which had nearly passed off, when she suddenly died, apparently from syncope. There was double optic neuritis. Post-mortem examination revealed thrombosis of the left lateral sinus and internal jugular vein and of some of the veins on the surface of the brain. No evidence of disease in the ear, bones of the skull, or meninges could be found, and there was nothing to suggest any cause for the thrombosis other than the anæmia. These are certainly very important and interesting cases, and they afford a possible explanation of the appearance of optic neuritis in some cases of anæmia. In the present case (No. 8), too, it is not difficult to imagine a thrombosis or resultant hæmorrhage so situated as to interfere with the function of one on both sixth nerves.

The occurrence of double optic neuritis in young women has been attributed to menstrual and uterine derangements, but the pathology of the combination is quite obscure. In connection with such cases, Dr. Bristowe's experience should not be forgotten. In a case reported by Broadbent,¹ in which headache, vomiting, and double optic neuritis had developed on sudden arrest of the menses, death ultimately occurred, and the post-mortem examination showed neither tumour nor meningitis—"the only morbid appearance in the brain was effusion into the ventricles." A second case, apparently of the same order, lost all her symptoms on re-establishment of the menstrual flow, but was blind from optic atrophy.

The eighth case of the present series is certainly not a riddle easily read, but it is at least a demonstration that double optic neuritis, even when combined with an event so suggestive of intracranial disease as paralysis of a cranial nerve, does not necessarily mean a diagnosis of tumour, or at least that such a combination is not inconsistent with a rapid and practically a complete recovery. But how is such a case to be distinguished at the outset from cases such

¹ *Transactions of the Ophthalmological Society*, 1880-81, vol. i., p. 108.

as the earlier members of the series here submitted. For if double optic neuritis and one such symptom as headache (Case 2), or vomiting (Case 3), is sufficient to justify a diagnosis of cerebral tumour, or, at least, to demand a very anxious prognosis, the same must be true of double optic neuritis when associated with such an event as a sixth nerve paralysis. The fact seems to be that there is no absolutely sure ground on which to base a distinction other than that provided by an exact knowledge of the progress of the case. When there is double optic neuritis there is ground for anxiety; whether it means definite organic disease within the skull or not it is in many cases difficult to say. But it is certain that even when there are other suggestions of cerebral disturbance the issue may be most satisfactory, whilst, on the other hand, the neuritis may stand almost alone as a sign of such disturbance, and yet the patient's eyesight, or even his life, be in great peril. In individual cases there may be particular features which will help the distinction between the two classes thus defined, but, on the whole, it is from a study of the development and progress of the case that the most reliable guidance will be obtained. How different, for example, is the character of the movement in Case 8 from what occurred in the earlier members of the series. It is not the mere existence of optic neuritis, of headache, of vomiting, etc., that gives cause for alarm, so much as the persistence, aggravation, or expansion of any one or combination of these symptoms. Further, the knowledge of the progress of the case on which an estimate and outlook are to be based must not be too limited in its range, for there can be no doubt at all that the urgency of the symptoms in many cases of cerebral tumour varies from day to day in the most remarkable manner. In Annie K. (Case 1) this was repeatedly seen; every now and then during the time when a fatal termination seemed probable, a day or two would occur in which pain and vomiting would entirely cease. "You go to a patient one day," writes Hughlings Jackson,¹ "and find him curled up in bed suffering intense pain, and hear that he has been vomiting urgently. Next

¹ *Medical Times and Gazette*, 1873, vol. ii., p. 541.

time you go to see him you find him sitting by the fire reading the newspaper, apparently quite well. *Do not be deceived by these pseudo-recoveries.*" It is a steadily continued, rather than a sudden, diminution of the symptoms which justifies a lessening of the anxiety necessarily born of the discovery of double optic neuritis. This is well seen in Case 9, where there existed double optic neuritis with such a large amount of swelling that, taking the ophthalmoscopic examination alone, the diagnosis of tumour seemed almost inevitable. No doubt the complete absence of all other cerebral symptoms was a grateful sign, and the history—if it could be relied on—of very rapid loss of sight made one think, or rather hope, that some other interpretation was possible. But it was only the steady movement of the case in the right direction that lessened the anxiety of the prognosis and justified an expectation of still better things. What exactly the diagnosis here ought to be may possibly be a question, but the case emphasizes what has just been stated in regard to the questions of diagnosis and prognosis arising in connection with attacks of double optic neuritis. The diagnosis now suggested is that of retro-bulbar neuritis. No doubt such a condition usually affects one eye only, or occasionally one eye, and then after an interval, the second. There seems no reason why it should not attack both eyes at one and the same time. Again, the diagnosis of retro-bulbar neuritis usually includes no appreciable early ophthalmoscopic signs, or only evidences of slight neuritis. But the evidences of papillitis certainly vary widely, and here at the worst it is a matter of degree. The rapid and early loss of sight, the history of pain in the eye-balls, the condition of the pupil light-reaction, the fact of nearly complete recovery, and the restoration of the periphery of the visual fields before central vision was possible, are all in favour of a retro-bulbar lesion. That both nerves were affected at the same time, and that there was also considerable swelling of the optic discs, are interesting features which give individuality to the case. The rapid failure of vision in this case may be usefully compared with the gradual decline of sight in the cases regarded as dependent on organic intra-

cranial disease—probably tumour. In these latter cases it is not as a rule during the stage of swelling and exudation that sight is prejudiced, but rather when the inflammatory movement is subsiding and the newly framed fibrous tissue, gradually becoming more compact, is by pressure injuring the vitality of the nerve elements.

It is of course a matter of common knowledge that double optic neuritis, as in Case 8, may exist without any complaint of defective sight and without any recognized failure when the usual tests for the visual acuity are applied. This was taught by Hughlings Jackson more than thirty years ago. The present cases show three possible conditions of the visual acuity with double optic neuritis. First, vision may be practically unaffected during the whole course of the neuritis (Case 8); it may be almost or completely lost during the stage of exudation and yet may subsequently be entirely regained (Case 9); or, as in the majority of cases, though it may be little or not at all affected during the acute inflammation, it may in the end be entirely destroyed (post-neuritic atrophy).

The diagnosis in all these nine cases may possibly be questioned. As has been already suggested it may be asked whether any of the first seven is really a case of tumour of the brain. On the other hand, it may be urged that Cases 8 and 9 ought, equally with the earlier members of the series, to be placed under that diagnosis, and that they must be regarded as differing from the earlier cases only in presenting slighter clinical evidence and a more complete recovery. It may be impossible to maintain an absolutely confident negative to this latter proposition, just as it is impossible—in view of the fact that occasionally a tumour of the brain is found when no evidence of its presence existed during life—to be quite certain that any particular individual, though apparently in perfect health, may not be the subject of some intracranial growth. One must accept what seem to be the reasonable probabilities of the case. Thus, when in children and young adults (as in Cases 8 and 9) the evidence of intracranial disturbance other than optic neuritis is little or none, when there is relatively rapid subsidence of the optic neuritis

and of any other existing symptom, and when also during such evidences of improvement no fresh symptoms emerge, it is justifiable to regard the optic neuritis as probably non-indicative of tumour, and to speak of the prospects of the patient in fairly confident and hopeful terms. But when the conditions attending double optic neuritis are the opposite of those just stated, the existence of gross organic disease is highly probable if not absolutely certain, and the risks to life, or alternatively to sight, are considerable. Some assistance to prognosis, if not also to diagnosis, is gained by treatment, and if under complete rest, mercurial inunction, with potassium iodide—or in the case of anæmic girls, iron—internally, the symptoms whatever they be do not show with a fair degree of promptitude decided improvement, the probability of organic disease is great, and the prognosis is correspondingly anxious. The differential diagnosis of tumour and meningitis must then be discussed, and in the event of the former being accepted the question of surgical interference at once arises. Unless there are evidences of the exact locality of the tumour, operation with a view to its removal could hardly be proposed, and statistical evidence shows very decidedly that in the great majority of cases of intracranial tumour removal is out of the question. Further, any proposal for an operation must be considered in the light of the fact that in a certain number of cases patients make a good recovery, with or without impairment of sight, under medicinal treatment, or, indeed, with no treatment at all; it must also be recollected that there may be more than one tumour, a state of matters not uncommon in young patients. The practical summing up seems to be that when optic neuritis, with or without other symptoms, is present in children or young adults, such medicinal measures as have already been indicated should be energetically applied. Should these fail and there exist localizing symptoms, as, *e.g.*, attacks of Jacksonian epilepsy, the possibility of surgical removal of the tumour comes seriously into view. In the absence of localizing evidence and the failure of medicinal measures, it is a fair proposal to trephine the skull with a view to relieve symptoms. Undoubtedly

headache and optic neuritis may be relieved in this way, and the patient may thus be rendered more comfortable even if his life is not prolonged. If sight is to be benefited by trephining, the operation must not be too long delayed; it must be undertaken during the exudative stage and before the nerve fibres have been structurally damaged by the pressure of the newly organized fibrous tissue.

There is one other practical point which may be noted. It arises from the possibility of optic neuritis being associated with, or perhaps due to, menstrual or uterine disturbances. Such cases as those reported by Broadbent¹ demonstrate this possibility, and it is supported by various authorities.² Hence when the patient is a female, and especially if there is absence of normal menstruation, attention to this function ought to be included in the treatment. The coincidence of suppression of the menses with optic neuritis must not however lead too confidently to the conclusion that the one is the cause of the other and to a correspondingly cheerful prognosis. In Case 1, in which the clinical evidence of tumour is complete, the patient ceased to menstruate at a very early stage of her illness, and the function for a long period remained in abeyance. Presumably the general functional disturbance resulting from the tumour interfered with menstrual activity just as it did with other of the bodily functions. Thus whilst it may be said there is evidence to support the view that optic neuritis may in some way or other result from menstrual irregularity, it must also be allowed that a tumour of the brain may in some indirect manner lead to suppression of the menses.

¹ *Transactions of the Ophthalmological Society*, 1880-81, vol. i., p. 108.

² *Ibid*, 1880-81, vol. i., p. 61.

TABLE I.

CASES OF TUMOUR OF THE BRAIN WITHOUT SYMPTOMS OR WITH SYMPTOMS OF ONLY DOUBTFUL SIGNIFICANCE.

Nature and Position of Tumour.	Author.
Cancerous tumour of greater part of anterior lobe, secondary to cancer of the lung. Nothing cerebral observed except manner at times peculiar and replies to questions "odd."	Hughlings Jackson, <i>Medical Times and Gazette</i> , 1873, vol. ii., p. 195.
Tumour of each lobe of cerebellum. No disturbance of gait or other motor symptoms.	Hughlings Jackson, <i>Ibid.</i>
Psammo-sarcoma, measuring 2 by $1\frac{1}{2}$ inches, growing from falx at junction with tentorium and compressing corpus callosum.	Goodhart, <i>Pathological Society's Transactions</i> , 1885-1886, vol. xxxvii., p. 16.
Mixed-cell sarcoma, of considerable size, in ascending parietal and supra-marginal convolutions; also a smaller tumour in occipital lobe.	D'Arcy Power, <i>Ibid.</i> , p. 54.
Myxo-chondroma, measuring $1\frac{1}{2}$ inches in longest diameter, springing from the falx and loosely embedded in a depression on the median aspect of ascending parietal convolution.	Hadden, <i>Ibid.</i> , p. 71.
Carcinoma, $1\frac{1}{2}$ inches in diameter, affecting dura mater and skull over right orbit. A prominence was observed over the right orbit but no symptoms of cerebral disease.	F. Taylor, <i>Ibid.</i> , p. 72.
Psammoma (two), each $\frac{1}{2}$ inch diameter, springing from choroid plexus of each lateral ventricle and occupying symmetrical portions of each descending cornu.	Hadden, <i>Ibid.</i> , p. 74.
Tumour, size of Tangerine orange, in occipital and temporo-sphenoidal lobes.	Hale White, <i>British Medical Journal</i> , 1886, vol. i., p. 117.
Tubercular mass in cerebellum.	Hale White, <i>Ibid.</i>
Tumour in frontal lobe. Only symptom, headache, until shortly before death, when optic neuritis appeared. Death sudden.	Hale White, <i>Ibid.</i>
Extensive tumour of orbital convolutions, destroying one olfactory nerve and producing considerable depression in bone. No cerebral symptoms unless "shortness of temper."	Alexander, <i>Liverpool Medico-Chirurgical Journal</i> , 1888, p. 253.
Large spindle-celled sarcoma in substance of hemisphere. Patient had occasional fits for ten years—controlled by potassium bromide—but no optic neuritis or other evidence of tumour.	Hadden, <i>Brain</i> , 1888-1889, p. 523.
Considerable cystic tumour in anterior part of temporo-sphenoidal lobe. Patient for several years under observation for "epileptic mania." No other evidence of tumour.	Trowbridge, <i>Journal of Nervous and Mental Diseases</i> , 1891, p. 217.
Large tumour of pre-frontal lobe. Optic neuritis for a long time, no other symptom until thirty hours before death.	Schweinitz, <i>Journal of American Medical Association</i> , 1893, vol. xxi., p. 607.
Numerous tubercular tumours both in sensory and motor regions. Patient, a child, quite free from head symptoms until a day or two before death, when some evidences of meningitis.	Middleton, <i>Lancet</i> , 1893, ii., p. 137.

TABLE II.

CASES OF RECOVERY, MORE OR LESS COMPLETE, FROM SYMPTOMS OF BRAIN TUMOUR.

Symptoms.	Sequel.	Author.	Notes.
Girl, 11 years, headache, vomiting, optic neuritis.	Recovery complete except blindness.	Hughlings Jackson, <i>Med. Times and Gazette</i> , 1865, vol. i., p. 626.	Patient unable to stand but could walk well with very slight guidance.
Woman, 19 years, headache, paresis of abducent and facial nerves on each side, with double optic neuritis.	Four months after, free from pain and generally well, but some degree of strabismus and facial paresis.	Wilks, <i>Brit. Med. Journal</i> , 1870, vol. ii., p. 61.	The diagnosis of tumour suggested but with some hesitation.
Man, 23 years, intense headache, convulsive seizures, optic neuritis.	Recovery to extent of walking about ward and reading newspaper, then sudden death (hæmorrhage from gliomatous tumour).	Hughlings Jackson, <i>Med. Times and Gazette</i> , 1873, vol. ii., p. 195.	Illustrates risk that attends even "recovery" from cerebral symptoms (see page 117).
Woman, 59 years, severe paretic and mental symptoms followed by headache, vomiting, and optic neuritis.	Illness extending over 5 months interrupted by periods of marked improvement—one of these lasting for 3 weeks. Death: glioma in white substance of posterior lobe.	J. B. Yeo, <i>Brain</i> , 1878, p. 273.	
Woman, 23 years, probable signs of tumour of cerebellum.	A year later general improvement, also some improvement in vision.	Gowers, <i>Trans. of Ophthalmological Society</i> , 1881-82, vol. ii., p. 34.	Paresis of upward movement of eyes was a special feature. Tendon-jerks exaggerated.
Woman, 19 years, extreme double optic neuritis with perfect vision for five months.	Then rapid and complete failure of sight, with optic atrophy—no other symptoms.	West, <i>Ibid.</i> 1882-83, vol. iii., p. 136.	Patient had been subject to chlorosis.
Headache and optic neuritis.	Recovery under administration of iron.	Hale White, <i>Brit. Med. Journal</i> , vol. i., p. 117.	Stated to be a case of anæmia.
Woman, 17 years, diplopia, strabismus, optic neuritis, weakness of lower limbs, absence of knee-jerks.	At end of six months quite well, vision normal. Recurrence of symptoms after 18 months, but again recovery.	James Anderson, <i>Ophthalmic Review</i> , 1886, vol. v., p. 121.	

TABLE II.—*Continued.*

Symptoms.	Sequel.	Author.	Notes.
Woman, 18 years, headache, vomiting, tinnitus, optic neuritis. Patient anæmic; nosyphilis.	Sodium iodide and mercurial inunction ordered. Patient completely well in two months. Six months later reported, "general health excellent, vision perfect."	James Anderson, <i>Ibid.</i> , p. 126.	
Man, 36 years, headache, hemiplegia, delirium, coma, optic neuritis.	Recovery complete under mercuric chloride and potassium iodide.	Suckling, <i>Birmingham Medical Review</i> , 1889, p. 159.	Recognized by author as probably a case of gumma though no history or evidence of syphilis.
Woman with headache, vomiting, marked optic neuritis. No history or evidence of syphilis.	Recovery complete under use of potassium iodide.	James Taylor, <i>Lancet</i> , 1894, vol. i., p. 133.	Author expresses his view that success of treatment is no proof of syphilitic causation.
Young man with headache, vomiting, intense optic neuritis, and epileptiform attacks.	Recovery apparently complete; two years later reported in perfect health.	Ransom, <i>Brain</i> , 1895, p. 534.	Renal disease, syphilis, plumbism, could all be excluded.
Woman, 39 years, headache, diminished mental power, convulsions, optic neuritis; no evidence or history of syphilis.	All symptoms disappeared under mercury and potassium iodide. For many years health good though occasional return of symptoms in a slight form and always yielding to treatment.	Althaus, <i>Trans. of the Clinical Society</i> , vol. xxxix., 1896, p. 39.	
Boy, 7 years, headache, vomiting, optic neuritis, paresis of left oculo-motor nerve, knee-jerks absent.	Disappearance of all the symptoms except blindness from optic atrophy. Fifteen months later general health reported to be good.	T. K. Monro, <i>Glasgow Medical Journal</i> , 1896, vol. ii., p. 173.	
Man, 63 years, history of severe head symptoms at 16 followed by blindness; knee-jerks normal.	Good health until shortly before death from cancer of stomach. P.M. = myxomatous tumour in cerebellum.	T. K. Monro, <i>Ibid.</i> , p. 176.	

TABLE II.—*Continued.*

Symptoms.	Sequel.	Author.	Notes.
Woman, 16 years, serious illness of three to four weeks' duration, severe headache, vomiting, optic neuritis, and ocular paresis. Temperature sub-normal, pulse slow, knee-jerks almost abolished.	Recovery complete except for very serious defect of vision (optic atrophy).	Alexander Napier, <i>Ibid.</i> , p. 372.	Author regards the case as one of tumour complicated by a transient attack of meningitis.
Girl, 8 years, serious illness with headache, vomiting, constipation, and loss of vision.	When seen at 17, well except for blindness and slight paresis on one side—occasional epileptic seizures since puberty.	T. K. Monro, <i>Ibid.</i> , 1897, vol. ii., p. 367.	
Woman, 17 years, some months of headache followed by vomiting and double ptosis; optic neuritis on examination and diminished knee-jerks.	Improvement for some weeks, then relapse, followed by almost complete recovery. Vision said to be "good" but evidence of post-neuritic atrophy.	T. K. Monro, <i>Ibid.</i> , p. 369.	

Mr. Hutchinson's cases of double optic neuritis in children associated with severe illness and various head symptoms, and followed by recovery except for partial or complete blindness, are recorded in the *Royal London Ophthalmic Hospital Reports*, vol. v. (1866), p. 307, and vol. ix. (1879), p. 124. In the same publication, vol. vi. (1869), p. 43, Mr. Hutchinson reports the case of a patient who suffered from pain in the head, ptosis, convulsions, and sudden blindness, and who in the course of six weeks, though still blind, was otherwise quite well. The same author [*Reports*, vol. xii. (1889), p. 65] has described two cases of double optic neuritis occurring without explanation in young men; one of the patients was seen eleven years later when he was the subject of double optic atrophy, but his general health was quite undisturbed.

Whatever may be the explanation of these cases, they manifestly have a very close clinical relationship to those recorded with this paper. The earlier ones afford abundant confirmation of the statement that double optic neuritis even when associated with severe cerebral disturbance by no means demands anything like a hopeless prognosis as regards the life of the patient, though the risk of permanent damage to vision is great. The last two cases further show that this danger to vision may also exist even when no cerebral events accompany the optic neuritis; in the parallel case (No. 9) of the present series the patient regained almost perfect vision.

Further cases illustrative of a capacity for recovery from severe symptoms of cerebral disease will be found in the late Dr. Bristowe's *Clinical Lectures on Diseases of the Nervous System*. And of course on all points concerning the diagnosis and prognosis in cases of intracranial tumour invaluable assistance is to be gained from the several papers of Dr. Hughlings Jackson. See e.g. *Royal London Ophthalmic Hospital Reports*, 1863-65, vol. iv., p. 389 *et seq.*; *Medical Times and Gazette*, 1873, vol. ii., p. 139 *et seq.*; *Transactions of the Ophthalmological Society*, 1880-81, vol. i., p. 60; and *British Medical Journal*, 1888, ii., p. 59.

CASE 1.

Severe illness characterized by symptoms of cerebellar tumour and followed by recovery except for total loss of sight (optic atrophy). Return of symptoms after two years' interval of good health, with subsequent complete convalescence. Patient reported free from all symptoms, except blindness, six years after first illness.

Annie K., æt. 16 years, admitted to Glasgow Western Infirmary July 28, 1893, complaining of vomiting, pain in the forehead, and unsteadiness of gait.

She enjoyed good health until two months before admission, when vomiting began, at first only on rising in the morning, but afterwards also throughout the day, both immediately after food and in the intervals between meals. In a week or so frontal pain became troublesome; the degree of it has varied much, but it is always present to a greater or less extent. She has been told that her eyes have recently become more prominent, the amount of protrusion varying from time to time. Her sight has been so bad that on several occasions she has run against objects in the house and street. Still, three weeks ago she went out to domestic service, though she soon found herself unfit for this, chiefly, it would seem, on account of her defective sight. Shortly after returning home she began to be very giddy at times; to stagger, with an inclination to fall backwards, when walking; and more recently she has complained of pain in the right side of the neck. Menstruation, which previously had been regular, has ceased during the last two months.

Patient is a well-nourished, healthy-looking girl. She is somewhat lethargic, and is slow in answering questions, but quite intelligent. There is marked retraction of the head. When assisted to rise she becomes giddy, and tends to fall backwards, indeed she would fall unless supported.

The pupils are equal and contract fairly well both to light and in convergence. In each fundus there is marked papillitis; visual defect is considerable; no squint or nystagmus; possibly

slight exophthalmos on each side. No tenderness on percussion of the skull. The superficial reflexes are normal; each knee-jerk is very marked—almost certainly exaggerated; cutaneous sensibility seems everywhere normal, and there is no paralysis of the limbs.

Thoracic and abdominal viscera normal to physical examination, and urine normal except for some phosphatic excess.

The sense of smell is very defective; she has observed this herself. Hearing not definitely abnormal.

The condition of patient during her first month of residence varied very much. Occasionally she would seem fairly comfortable, but for the most part frontal pain was extreme and vomiting very obstinate. At times she passed into a semi-comatose condition and appeared very likely to die. The bowels could not be evacuated without enemata, and unless the catheter was regularly used the urine was passed in bed. Temperature almost invariably subnormal; pulse from 60 to 72. The swelling of each optic papilla became less, the light response of the pupils diminished, and the vision was reduced to mere perception of light. Tested on several occasions the knee-jerks were found to be absent.

On August 23rd some evidence of right facial paresis was observed, the upper as well as the lower muscles being involved; general condition somewhat improved.

On the 2nd of September she somehow or other fell out of bed, but apparently did not hurt herself. The nurse, however, noticed that during the night she did not move her left leg, and on examination the limb was found absolutely paralysed. The knee-jerk at this time was quite decided on each side. She was now again very ill, the retraction of the head very marked, mental faculties much obscured, and urine repeatedly voided involuntarily.

By the middle of September she was much better again, frontal pain and vomiting much less, retraction of head much less, power returning to left lower limb, and no advance of facial paresis. On the 19th the knee-jerk was found absent from the left side, and only a trace present on the right.

There were one or two occasions during November when

pain in head and vomiting were troublesome, but on the whole the patient made decided progress. She gained flesh and became bright and cheerful, though she was quite blind (optic atrophy); there was also marked horizontal nystagmus. The functions of the bowels and bladder became normal, and apparently complete voluntary power was regained over the left lower limb; the evidence of right facial paresis had also receded, and the sense of smell somewhat improved.

When she left the hospital on December 4th she could stand steadily and without giddiness, and could walk with very slight assistance. Examination showed a very marked left knee-jerk, the right being both slight and sluggish.

The most impressive feature of this case was the recovery of the patient after her condition for many weeks had seemed practically hopeless. The facts of the case show the diagnosis of intracranial tumour "writ large," and evidence is not wanting to show that the situation of the tumour is probably the right hemisphere and middle lobe of the cerebellum. A combination in aggravated form of vomiting, vertigo, and optic neuritis is in itself, though not conclusive, sufficient to suggest a cerebellar growth; marked retraction of the head, and possibly nystagmus, are also not without significance in the same direction. The difficulty in maintaining equilibrium and the tendency to fall backwards indicate disturbance of the middle lobe. Paralysis of the left lower limb associated with right facial paresis show that the pressure of the growth is mainly towards the right side, so as to involve part of the motor tract above the decussation, and the trunk of the facial nerve. This is in harmony with the usual experience of paralyses produced by pressure of a laterally situated cerebellar growth. It is noteworthy that the headache was entirely frontal, showing how little reliance is to be placed on the site of the pain as indicative of the situation of the tumour. The varying character of the knee-jerks and the presence of anosmia—a rare result of intracranial tumour—are also special features of the case.

Patient was again under observation from October 10th to November 27th, 1895. In the interval she had for the most

part enjoyed good health, but, recently, frontal pain, vomiting, and giddiness had troubled her. Under rest in bed, etc., these symptoms subsided, and she left the hospital in good general condition, and with no disturbance of gait other than might well be due to her blindness. Knee-jerks *in statu quo*. In June, 1899, patient's sister writes: "A. is never very strong, but she is free from her old symptoms, except the blindness."

The risk of recurrence of the symptoms after even a considerable period of quiescence, and the possibility of a second "recovery," are thus further features of clinical interest in the record of this case. (See also Case 5.)

CASE 2.

Absolute blindness, with marked papillitis and subsequent optic atrophy in right eye (and presumably also in left), with history of severe pain in head, and, later, disappearance of knee-jerks; patient seen in good general health eighteen months after onset of symptoms.

Martin T.,¹ æt. 10 years, complaining of defective sight and pain in head, May 1, 1898. Present illness commenced ten months ago. Pain in the head has at times been very severe, apt to come on in the evening and keep patient from sleeping. The loss of sight has been of gradual development. At no time has it been necessary to confine patient to bed, and there have been no convulsions and no complaint of giddiness. Vomiting has occurred occasionally, but always after taking milk. No history of a fall or blow on the head.

Patient is a pale, delicate-looking boy; general physiognomy not suggestive of hereditary syphilis; he is almost absolutely blind. The left cornea is hazy from interstitial keratitis, and evidences of the same condition exist in the right eye. The pupils show very defective light response. In the right fundus there is marked papillitis which is showing signs of retrogression. Midway between the disc and macula is a small patch of choroidal atrophy, and to the inner side of this

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is a group of yellowish-white, highly refractive dots. The left fundus cannot be seen on account of the corneal haze, which, however, is not sufficient to account for the extreme visual defect.

Patient walks well and quite without ataxia; knee-jerks present but almost certainly defective. Superficial reflexes present except plantar. No evidence of disease in thoracic and abdominal viscera; urine free from albumen; some enlargement of cervical lymphatic glands.

Family History.—Father and mother alive and well; mother lost a sister from consumption. Patient is youngest of a family of eight; of these two died of “convulsions,” one with “something in her brain” and one—the first-born—at seven months, cause unknown. The other children are said to be healthy (two examined show no evidence of hereditary syphilis).

Patient was kept under continuous observation for two months. At times he complained of pain in his head and displayed a very violent temper, but his appetite was good, he never vomited, and his general nutrition improved. Examined repeatedly during this period the knee-jerks were completely absent. The only other change was the advance of optic atrophy in the right eye; the details of the left fundus could never be seen. In the beginning of December, 1898, the boy was again seen; he seemed quite cheerful and fairly vigorous. Blindness was absolute. Knee-jerks absent.

Presuming that this is a case of intracranial tumour, the only indication of the site of the growth is afforded by the loss of the knee-jerks—a fact which points with some degree of directness to the cerebellum. The probabilities are that the tumour is tubercular in nature even though the boy is undoubtedly the subject of hereditary syphilis. Syphilitic tumours of the brain are not common in children whereas tubercular growths are frequent, and the cerebellum is their most usual site. The family history seems distinctly tubercular. The absence of evidence of tubercle in any other part of the body—unless possibly the cervical lymphatic glands—does not mean much in a child, though in adults, according to

Gowers, "the signs of phthisis are rarely absent when there is tubercular tumour of the brain." Further, the boy appeared to receive little or no benefit from antisiphilitic treatment, whilst he improved considerably under the use of iodide of iron and cod liver oil.

CASE 3.

Double optic neuritis followed by blindness and optic atrophy; history of vomiting, with slight headache every morning for six months preceding loss of sight, but no other cerebral symptoms and no "illness" confining patient to bed even for a single day; knee-jerks present, though right possibly defective.

Jas. C., æt. 13 years; examined on April 25, 1898. His complaint is loss of sight. According to the father, the boy had perfectly good sight until the early part of January, when he fell in the street and struck the back of his head against the pavement. No apparent injury followed the fall, but in the course of a fortnight the boy complained that he "could not see well," and soon he appeared to become quite blind. It seems, however, that for six or seven months before the fall the patient was troubled with vomiting, which occurred daily, but only in the morning before breakfast. After each act of vomiting he had some pain in his forehead for a time, but he was always able to go out and distribute newspapers and then to return and take breakfast, and afterwards to go to school. The vomiting still continues, but is entirely confined to the early morning. At no time has the boy been confined to bed or regarded as seriously ill, and as a proof of the comparatively recent failure of his eyesight it is mentioned that he won a prize for drawing at Christmas. It is solely the blindness which, in the view of the father, constitutes the lad's disability.

The boy has not even perception of light in either eye; the pupils are dilated, and show no light response; each fundus provides evidence of consecutive optic atrophy.

He is a fairly vigorous-looking boy, quite free from evidence of hereditary syphilis. There is some enlargement of the submaxillary lymphatic glands, but no evidence of disease in the thoracic or abdominal viscera.

Knee-jerk present on each side, the right not being easily provoked and less distinct than the left (confirmed at a later date); no ataxia in gait.

Family History.—Of three children born before patient two died from “suppressed measles,” the third being alive and in good health. Since the birth of patient there have been eight pregnancies, all terminating at full time—three of the children survive; the other five all died when about eight months old from “wasting”—diarrhoea, vomiting, and swelling of belly. Father and mother in good health, and with reputed good family records.

There is nothing in this case to definitely indicate the site of the tumour. Perhaps all that can be said is that the family history is marked by a number of events almost certainly tubercular, and that the most frequent situation of tubercular tumours is the cerebellum. Some difference between the degree of the knee-jerks can hardly be claimed as of definite diagnostic value. The principal value of the case is the demonstration it affords of the great importance of giving the most careful attention to any symptom of possibly cerebral origin, and more especially if such a symptom—even though existing alone—is extended over a considerable interval of time. It is practically certain that grave intracranial disease in this case produced for some considerable time no evidence of its existence other than an attack of vomiting, with some frontal headache, every morning. Then followed optic neuritis, which did not seriously affect vision until it began to pass into post-neuritic atrophy. Whether the diagnosis of tumour be justifiable or not, the practical importance of such a sequence of events as is here presented is obvious.

CASE 4.

Serious illness at 15 years, marked by frontal pain, vomiting, and failure of sight; four years later patient seen in good health, but the subject of post-neuritic optic atrophy.

Rose M., æt. 19, complaining of defective sight; examined August 7, 1898.

The failure of sight is referred to an illness which confined her to bed for six weeks in the summer of 1894. The symptoms of this illness were obstinate vomiting, pain in the forehead, failure of sight, and general weakness. The medical man in attendance (now deceased) is reported to have said that she had a tumour of the brain. Gradually these symptoms subsided, but the sight, though it improved somewhat, soon reached the condition in which it is at present. No history of injury to the head. Menses have always been somewhat irregular; she cannot say there was anything unusual in this respect at the time of her illness four years ago.

Patient is a tall, healthy-looking young woman. R.V., counts fingers at 6 feet, and reads J. 16 at 8 inches. L.V., hand movements only.

Each pupil is dilated, and exhibits only a feeble light response. In each fundus the disc is white, with "fuzzy" edges, arteries small, and vessels at centre of disc concealed by a filmy haze. There are distinct "white lines" along many of the vessels; at the left macula there are fine retino-choroidal changes. The knee-jerks are distinct, and the gait free from evidence of ataxia. Cardiac sounds and pulmonary physical signs normal: some, but by no means marked, evidence of anæmia.

Family History.—Father and mother alive, and in good health. Patient is fourth in a family of ten—one brother was killed in an accident, the others are all strong and healthy. She has lived all her life in a small village in the west of England.

The history and existing post-neuritic atrophy show the

three symptoms, viz., headache, vomiting, and optic neuritis, which, to quote Hughlings Jackson, "make the diagnosis of an 'adventitious product' within the skull almost certain." That the facts justify such a diagnosis in the case of a patient apparently exceptionally favoured as regards hereditary and other influences seems most unaccountable. The pathetic spectacle of a seemingly healthy and vigorous girl reduced to hopeless blindness may well emphasize the urgency of every possible measure which may conceivably help to avoid so disastrous an issue.

CASE 5.

Acute evidence of intracranial disease of three months' duration in boy of 7 years, followed by recovery, with the exception of blindness (optic atrophy) and loss of knee-jerks; return of headache, vomiting, etc., with a fresh attack of papillitis after an interval of three years; repeated subsidence of all symptoms of active disease, and boy six months later known to be in good health.

Jno. D., æt. 10 years, seen in December, 1896. He is reported to be blind, but otherwise in good health. Some eighteen months ago he had a very serious illness, being confined to bed for nearly three months, and at times he was thought to be dying. The most troublesome symptom of this illness was vomiting; he also had pain in his forehead, his head was drawn backwards, and his sight failed. The diagnosis at the time was "tumour of the brain." He is now a plump cheerful-looking boy. He is quite blind, and the pupils have no light response; in each fundus there is decided evidence of a former papillitis, now in the atrophic stage. Knee-jerks absent, gait normal, memory and mental condition generally seem quite acute. Thoracic and abdominal viscera healthy.

The boy is said to have had a brick fall on his head many years ago.

July, 1898. The boy has been in good health until six weeks ago, when vomiting returned, accompanied by diarrhoea, and continued until within the last four days; there

has also been pain in the head (frontal); at times he has seemed to be unconscious, and on two occasions has had a "fit," in which he became "stiff," but not convulsed. He is now much better in every respect, and indeed he says he is quite well. The ophthalmoscope shows the addition of a recent papillitis to the atrophic condition formerly noted. Knee-jerks are absent.

In January, 1899, the boy is reported to be "stouter and more cheerful than ever." The family history shows a twin brother and three younger brothers and sisters alive and well. Father and mother also enjoy good health; no knowledge of family liability to chest or other disease.

This case, like Case 1, shows how liable the symptoms of intracranial disturbance are to return after even a considerable period of quiescence. Every care should therefore be taken to reduce the risk of this possibility by maintenance of the general health and by the use of such agents as cod liver oil, iodide of iron, etc.

There is one feature attending the recrudescence of the symptoms here which is very unusual, viz., a second attack of papillitis in discs atrophied as a result of the first attack. Gowers in his work on *Medical Ophthalmoscopy* writes, "If a disc has become completely atrophied it is very rarely again the seat of inflammation." Cases of second attacks of papillitis following complete optic atrophy have, however, been observed by Hughlings Jackson and by Schweinitz. "When atrophy is partial or absent, in rare cases two attacks of neuritis may occur" (Gowers).

CASE 6.

Blindness (post-neuritic optic atrophy) and occasional quasi-epileptic attacks, but otherwise good health, in a girl of 16 years, who two years before was for several months seriously ill with symptoms of active intracranial disease.

Ada L.,¹ æt. 16 years; examined on June 20, 1898. She complains of blindness. Patient is a delicate-looking girl.

¹ Seen in consultation with Dr. Jas. Smyth.

The pupils are dilated, and have only a slight response to light; they move freely in convergence. Each fundus shows evidence of a former neuro-retinitis with marked atrophy of the optic papilla. Blindness appears to be absolute. The cardiac sounds are pure; R.M. over lungs abundant and free from râle; nothing noteworthy in physical examination of abdomen.

The first note of the knee-jerks is that they are "present," subsequently the right one is "hardly obtained," and the left elicited only with difficulty. The gait is free from ataxia.

From May to November, 1897, she was very ill, suffering from attacks of giddiness, severe pains in the head (which was "drawn back"), and gradual failure of sight. In the course of this illness she had two "fits," attended by unconsciousness, and followed by "paralysis of one arm and leg." No history of injury to head or of discharge from either ear. With the exception of the defect of sight she has been improving since the beginning of the year, and is now able to walk about undisturbed except by an occasional pain in the head; there is no sign of hemiplegia. Her mother adds that patient at times has some kind of "fit" or "attack" which she cannot well describe. There is no loss of consciousness and no convulsion, but patient calls out "I am worse," and clutches hold of anyone who may happen to be at hand. Some of the attacks occur at night-time, and on several occasions there have been involuntary evacuations.

Family History.—Father and mother enjoy good health; the latter says a number of her relatives in the earlier generations died of consumption. There are three other children besides patient, and these are alive and well. No deaths; no miscarriages.

CASE 7.

Blindness and bilateral optic atrophy without evidence of previous neuritis or papillitis in girl of 21; other evidences of (presumably) intracranial disease being nystagmus, quasi-epileptic seizures, explosive temper, and absence of knee-jerks. History quite free from evidence of any acute cerebral attack, the loss of sight occurring quite gradually when patient aged 11 years, and unaccompanied by any other symptom of intracranial or other disease.

Edith H., æt. 21 years; examined on July 4, 1898. Her statement is that she is quite blind.

Until 11 years of age had good sight, and was in every respect strong and well. Then gradual loss of sight came on, and slowly progressed until in the course of twelve months or less she became quite blind. The defect was first noticed by the girl making mistakes when reading and tumbling over objects when walking. At no time did she complain of pain in her head or of vomiting. (Pressed on this point, she says that about a year ago she often used to vomit after breakfast, but with this exception she has never been troubled with vomiting.) She is sure that the failure in her sight was not accompanied by any general illness, and she was never confined to bed. No history of injury to head. For the last three years she has been subject to occasional fits in which she "gets stiff but does not fall, and is unconscious for a minute or so." She is liable to outbursts of temper. But her general health is and always has been very good; menses quite regular during last five years. Patient is a healthy-looking woman. She is quite blind, and the pupils which are widely dilated have no light response. The optic disc on each side is very white, the retinal arteries, and perhaps also the veins, diminished in calibre. Neither in the disc itself nor in the fundus generally is there evidence of a previous neuro-retinitis. There is marked horizontal nystagmus (present for five years). Knee-jerks absent; no ataxia in gait. Examination of chest and abdomen entirely negative.

Father, mother, three other members of family—older than

patient—have quite good sight and satisfactory health. No knowledge of fits, nervous disease, or defective sight in any relatives.

The reasons for placing this case as one of intracranial tumour have been advanced in the body of the paper (p. 111). The failure of sight without any other suggestion of intracranial mischief is remarkable. It is another example of how grave intracranial disease may for a long time produce only slight signs of its existence.

CASE 8.

Paralysis of sudden onset of right external rectus, also double optic neuritis, but no other evidence of cerebral disease, and no considerable visual defect other than diplopia; patient, a girl of 17 years, somewhat anæmic, but otherwise physical examination normal. Recovery complete and apparently permanent.

Maud E.,¹ æt. 17, cook, complaining of double vision (June 7, 1898). The diplopia is of a fortnight's duration; it was of sudden onset, a group of children at whom patient was looking "suddenly becoming double." She has no other ground of complaint. Patient has always been healthy, though admits some recent loss of colour, breathlessness on exertion, and unduly long menstrual intervals. She was menstruating at the time the diplopia developed.

There is almost complete paralysis of the right external rectus muscle; tested with a flame and coloured glass there is homonymous diplopia increasing to the right; she also recognizes increasing separation of the images to the extreme left, but there is no appreciable defect in the action of the left external rectus. No ptosis or other defect of ocular movement, and pupils equal and with normal reactions.

R.V. 6/6 ·5 Hm. J. 1 at 5 inches.

L.V. 6/6 ·5 Hm. J. 1 at 5 inches.

There is marked optic neuritis in each eye—the disc is moderately swollen, with numerous hæmorrhages on the

¹ Seen in consultation with Dr. D. O. Macgregor.

surface of the swelling; the vessels as they leave the disc are concealed by exudation, and the veins generally are enlarged and tortuous.

The visual fields for white and colours are normal, and there is no central or other scotoma.

Physical examination reveals nothing beyond a venous hum at the root of the neck and a blowing murmur with each expansion of the subclavian arteries. Urine free from albumen and sugar.

She has but an incomplete knowledge of her family history, all she can say is that her mother died of "consumption and heart disease."

Patient ordered to take complete rest, and iron prescribed.

21st June. General condition improved; papillitis somewhat less marked; she does not read 6/6 fully with either eye; paralysis of right external rectus less complete.

25th July. Improvement still more manifest. There is now only a slight degree of paralysis of external rectus. With the right eye she makes mistakes in reading both 6/9 and 6/6, and with the left does not read 6/6 fully. There are now definite changes at each macula, a circular area of a deep red colour and about the size of the optic disc occupies the centre of each fundus, and on this, more especially in the right eye, are a number of irregular, whitish, lustrous spots. Papillitis subsiding.

2nd August. The patient is looking very well. There is now hardly appreciable defect in the outward movement of the right eye, but homonymous diplopia can still be detected to the extreme right when the flame and coloured glass are used. The greater part of the margin of each disc can now be seen; fundi otherwise unchanged. Visual acuity as before; recognizes all colours in 1 mm. square in central vision.

30th August. Improvement continues. The discs are nearly normal and the congested areas in the macular regions less prominent. There is no diplopia even to the extreme right.

20th September. Patient regards herself as quite well, and appears to be so. The fundus on each side remains

as before, and there is slight defect in the visual acuity of each eye.

She is allowed to return to work; to continue to take iron for another three months.

30th March, 1899. In good health. Evidences of former neuritis readily appreciable in each fundus, but vision of normal acuteness.

The difficulties of diagnosis in this case have already been discussed (p. 118). As the patient was undoubtedly anæmic, it seemed right to order complete rest and the administration of iron. Had there not been decided improvement under this treatment it would have been wise to have tried mercurial inunction (see Case 9).

CASE 9.

Rapid failure of vision in girl of 14 years, with marked double papillitis not accompanied by headache, vomiting, or any other suggestion of cerebral disease; prompt improvement under mercurial inunction, the visual acuity reaching almost to the normal level within three months. Patient after an interval of nine months found to have nearly full vision, normal visual fields, and good general health.

Ellen D.,¹ æt. 14, seen on June 15, 1898, complaining of failure of sight in each eye. She could see quite well until a week ago; at that time she had a shooting pain in the right eye, and the sight of the eye rapidly failed. A similar experience soon occurred in the left eye. No pain in the head and no vomiting, and general health quite unaffected.

R.V., denies even perception of light. L.V., hand movements only. Pupils dilated: right no response to direct light, but contracts in consensual stimulation; left, considerable light response, but contraction not well sustained. No ophthalmoplegia externa. Right fundus shows decided papillitis (fully +3 D), with a number of hæmorrhages on the surface of the exudation; similar but less marked condition in left eye.

Movement of the eyes in any direction does not cause pain, and palpation of the globes scarcely causes complaint.

¹ Reported by permission of Mr. Poulett Wells, M.B.

Patient is well nourished; no evidence of anæmia; physical examination of chest and abdomen normal; urine normal. Knee-jerks normal, no ataxia in gait, superficial reflexes easily obtained, cutaneous sensibility and motor functions undisturbed. At no point is the skull tender to percussion. She has never been very strong, had rickets in infancy, jaundice four, and measles two years ago. No history of injury to head or of discharge from ears. She has never menstruated.

Family History.—Father alive and well; his mother and a sister and brother died of consumption; mother lost a brother from the same disease. Patient is the eldest of a family of four—one died of consumption, the other two are “delicate.”

6th July. Mercurial inunction has been used every night. She can now count fingers in the peripheral part of the field of each eye, but not with central vision, and can move about the ward without stumbling against objects as she did on admission. The papillitis has distinctly subsided.

6th August. Has been away at seaside. Vision still improving. R. counts fingers at three feet in central vision; L.=6/24. Contraction of pupils under light much more marked, but that of right is not well sustained. Discs white and filled in.

13th August. R.V.=6/18; L.V.=6/12 (less one letter).

3rd September. R.V.=6/9 (two letters); L.V.=6/9 nearly. Pupils not definitely abnormal.

17th September. R.V. 6/6 part; L.V. 6/6 part. Fields full, no central scotoma, recognizes all colours through an opening 1 mm. sq. Whiteness of discs very decided, and arteries look small.

June, 1899. Patient is in good health, and the visual acuity is almost, though not quite, up to the normal level. Objective examination of the eye negative, except for evidences of former papillitis.

It is suggested that this is a case of retro-bulbar neuritis affecting both sides, and with a greater degree of papillitis than is usual, and the reasons for holding this diagnosis are given in the body of the paper (p. 122).

ON EPILEPTIC SPEECH.

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THE speech faculty of the epileptic has hitherto received very little attention, though passing references to it have been made from time to time by several writers, viz. Kusssmaul, Ross, Wylie, and others. Wylie has stated the well-known fact that temporary aphasia appears sometimes as the "aura," sometimes as an immediate consequence of a fit. Kusssmaul confirms this, and Ross writes, "In some cases the warning of an epileptic attack consists of a sudden inability to speak, and it is very probable that word deafness and word blindness are by no means uncommon auræ." While saying so much, Ross admits what is certainly true, that motor aphasia is the more readily noticed, and, as obscuring the question of aphasic auræ, he admits the mental confusion attending the onset of unconsciousness, a factor of some importance. Bradylalia (slow speech) and echolalia (echo speech) have also been noticed by observers at home and abroad. They are, however, so frequently observed in developmental speech, and in other nervous and mental diseases that too much may be made of their significance.

The relation of emotion to speech is well illustrated in Bastian's work on *Aphasia* (p. 5), where there is described the case of a boy, the son of a leading barrister, who had been subject to "fits" at intervals during his early childhood. The first occurred at the age of nine months. They ceased at the age of two years, and the child appeared to be all right,

intellectually and otherwise, except that he could not talk. Before he was six years old, when an accident happened to one of his favourite toys, he exclaimed "what a pity," although he had never previously spoken a word. In the case of children of backward speech this has frequently been observed, and such must have come under the notice of not a few family physicians. The point is that the tardy mechanism which may have been making abortive attempts at speech for some time previously, succeeds at last owing to an accession of vocal energy. This accession of vocal energy is due to emotional excitement acting upon the respiratory centre, and exciting a deeper respiratory movement, which during expiration gives the larynx the necessary blast of air at the precise moment when the emissive energy of motor speech is discharged.

The innervation of the vocal speech mechanism requires to be allowed for, if we would adequately comprehend the physiology of the production of words and sentences, but this I pass by, merely observing that it is a factor of considerable importance in our study of epileptic speech. This innervation will be regarded here also in relation to emotional states, for the epileptic is a creature of moods and tenses in the highest degree, and his speech is thereby affected. It has been stated that aphasia is the condition which some observers have noted as the most usual speech affection of the epileptic; but aphasia is now a term of very comprehensive meaning, and the modern conception of the term holds within its limits certain varieties which we do not find associated with epilepsy. Moreover, dysphasia is a term which includes more of the speech affections of the epileptic than does aphasia. The distinctions which will be recognized here are—

- (a) *Aphemia*—inability to speak, depending on affection of the co-ordinating centre for the muscles producing articulate sound.
- (b) *Amnesia*—loss of the memory of words.
- (c) *Agraphia*—inability to write.

In the examination of the cases to be immediately referred to, the outlook for word deafness and word blindness was negative

in its results, but I do not dispute Ross's proposition, though when one considers the mental equation before and after seizures, his statement must be regarded as a difficult one to prove. The clinical study of the following cases was conducted while their intelligence was unclouded by the shadow of a seizure or its after stupor. They are men and women who have been insane for varying periods, some two or three years, others fifteen to twenty years. Naturally, we look for mental deterioration in the older cases, just as we see it in chronic mania or dementia, but in the latter the speech deterioration is mental rather than motor, the emissive or co-ordinating faculty is not impaired to anything like the same degree as in epilepsy. In the early stages of epilepsy—apart from insanity altogether—the speech affections are less marked, though early evidence of them may in some cases be manifest, especially bradylalia.

In considering the matter systematically, the following points were kept in view—(1) the mental state, distinguishing the emotional and the intellectual; (2) the receptive or subjective function of speech; (3) the expressive or objective function; (4) the vocal mechanism and its innervation; (5) the oral mechanism and its innervation; (6) variations in the individual. The patients were each interviewed on two separate occasions.

Case 1. M. B., age 47, insane eight years, education poor, memory for past and recent events impaired. She is capricious, easily roused, emotional instability is very marked, and her speech, which, in her placid moods, is low, slow, stuttering, and slurred, when her temper is roused becomes suddenly loud, fierce, denunciatory and free from stuttering, with staccato pauses. Then her attitude is tragic, her arms are raised with threatening gesture, her face is flushed, her chest heaves, and her voice is loud and resonant.

To every patient the first question was, Have you ever noticed any trouble with your speech? Some resented the idea; most of them at first denied the imputation. Epileptics usually deny that they have had a fit, and are very intolerant of the suggestion that anything is the matter.

With this explanation in view, the answers will speak for themselves. Letters, syllables, or words are spaced according to their cohesion to each other or want of cohesion. *Question:* Have you ever noticed any trouble with your speech? The answer comes in a jerky, spluttering stream of broken talk. Sometimes she stops short with a sudden "catch in her breath." Her reply to the question was as follows:—"Quite so—I de be—," then, as if to excuse her faulty speech, "Thir's yin o' my teeth kin' o' slack." Here she does not stop, but dribbles away in slow monotone, "doon to mel—ans—field—so will—be coming on—for—my—age just now,—my faither—would be coming to, so—came for him and my mother going together forty-four."

While speaking in this strain, it was noticed that the apparent incoherence was due to amnesia. She had a difficulty in getting hold of the right word, and, like a person who stutters and introduces irrelevant sentences to get out of a difficulty, she introduces words to excuse herself, and often makes confusion worse confounded.

The amnesia is not marked except when a proposition is made to her, or when a question is asked which requires the construction of sentences. If shown a key, watch, or knife, she names them correctly enough, but always cautiously, as if conscious that she might trip in with the wrong word, thus:—"Well—I would call it—a key," or "Well, I would say it is ca'd the knife—thing," or "It's a watch—if I would say it." She repeats the 23rd Psalm (metric version) correctly, and with very little trouble; but here the mental effort is less and the words do not need to be made up in sentences; these are ready made for her. When she is excited the voice is raised, and the words come more trippingly, though irregularly, the rhythm reminding one of the pulse beats of an irregular heart. At such times the end of the sentence is cut short from failure of breath owing to faulty vocal innervation. To sum up this case there is (1) amnesia—her vocabulary is very limited, and she very frequently puts in the wrong word. (2) Defect of articulation, stuttering, and explosive speech. The mouth in quiet speech, which is her usual when not excited, is almost

closed, the action of the jaws being feeble; this may be said also of the lips and tongue, which are by no means mobile, and which with the supra oral muscles are tremulous. (3) Deficient phonation; the respiration is shallow, and this may account for it, as, when she gets excited and the chest heaves the voice is much louder and articulation is more distinct. (4) There is marked bradylalia. Echolalia is sometimes present. There is considerable gesticulation when excited.

Case 2. W. J., æt. 30. Has taken fits since the age of 18. the exciting cause of the first being the passage of a tape worm. His expression is quiet and sad, but he is intelligent, and by no means devoid of humour. He is when free from fits quite reliable, and quite capable of giving intelligent answers to questions.

To the question, "Have you ever noticed any trouble with your speech?" he replied, "I've felt pretty far back in speech this time back: the language that comes from me is rather short of grammatical—feels as if there was a weight keeping back the words." All this is said very slowly and with apparent deliberation. "Do you feel a difficulty in getting the right word?" "It takes a long time to compose it," meaning the sentences. If excited, *i.e.* if there is any emotional disturbance, his reply is not quite so intelligible, as when the same question was repeated some days later, he thus replied, "For a long time—— education also to bring me up to satisfaction, so as that I wanted to keep myself as I intended at first." His voice breaks, there being vocal tremor, especially when he is emotionally roused. Before and after fits he is quite conscious of the fact that speech is more difficult. He observed, "Half an hour after when I come out of a fit, if any one spoke to me couldn't answer them." When asked if he was ever altogether speechless, he answered, "Well, I can't consider for that"; then a pause as if for breath, then the echo, "altogether speechless." There is no agraphia, no word blindness or word deafness, and he has no recollection of either of the two latter occurring as an "aura." His invariable "aura" is a sensation in the left arm and side.

Inspiration, even when asked to take a deep breath, is rather shallow, but during emotional stress his respiration is more active, and his voice is louder. We may therefore say that here there is (1) partial amnesia, (2) diminished phonation, (3) weak articulation, with tremors, and that according to his emotional state these vary. The labio-dental movements are certainly rather inert. In this case there is very little gesture; but that is exceptional, and even this man when excited, buttonholes one in a confidential way, rather usual with epileptics in their quiet moods.

Case 3. P. H., æt. 31. Insane five years. Ascribes first fit at the age of 15 to a fright. Had taken to smoking before then. The degeneration in this case is marked: he has shown considerable nervous failure in the last three years. It was noticed on admission that his speech was slow, thick, and indistinct, with an appearance as if he was swallowing some obstacle, after speaking each word. In his stuporose states swallowing is difficult, and he is very liable to choke. His vocabulary is very limited. Like not a few epileptics, he has stereotyped phrases which he invariably employs in certain given circumstances. His consciousness of amnesia has led him to adopt them rather than struggle to compose fresh sentences. Thus every morning and evening at the medical visit he receives the superintendent and others with these words, holding out at the same time his right hand for a shake, "How—do—you do—Dr.—Clark—and Dr.—Kerr—and Mr.—Campbell—and Nurse—Thomson—and—my respects—and—I'm quite well."

He frequently repeats the words of questions put to him, as if to give him time to jog his memory and stimulate recollection. Instinctively he seems to feel that echolalia by its sensory stimulation of the auditory centre may rouse recollection. Bradylalia here is very marked, but much less noticeable under emotional excitement. When asked if he had any difficulty of speech, he replied, "Sometimes—I am—very well at it (*i.e.* getting the right word) some days I am—not very sure—of myself—and I stop—but if—word is ready—and if—difficulty is in mouth—big—words—I can't

say." There is not merely amnesia, but aphemia. Even when he knows what he wants to say there is a difficulty owing to obstruction in the speech mechanism. He explains this by speech and action—"There is" (as he puts his hand to his throat) "a diffi—culty as if stopped in the throat."

He explains further that crabbedness (rise of temper) sometimes makes him use the wrong words, and here again emotional disturbance shows its effect not merely on the speech mechanism, but on memory itself.

There is slowness in answering, when questioned as to the names of objects, his explanation being that he is afraid of saying the wrong word. Feeling his ankle, which has been sprained, and is still swollen and stiff, he says, "It's more stronger,—it's more stronger" (echolalia). He seems to feel that pantomime helps him along, and probably this is why epileptics are often demonstrative and gesticulate so much.

As regards oral and vocal speech, there is the same lack of innervation, the same drawling, stuttering speech already described, and the voice is low and respiration shallow. Not only is there interruption of the speech current, but there is incoordination of the laryngeal (vocal) and oral mechanism. There is frequent tremor of the lips when speaking.

Case 4. R. N., æt. 60. Insane twenty-two years. Epileptic for forty years due to injury in a mine (wound on temple) and probably fright. A hypochondriac, but a most violent patient at times. There is less to notice about his speech than in some more recent cases. There is no agraphia, word blindness or word deafness, but there is amnesia, and his speech is sometimes slow, hesitating and tremulous. He is very emotional, and this affects his voice. Asked if he ever noticed anything wrong with his speech, he replied, "Ne—ver noticed anything wrong with my speech" (echolalia), but later admitted when "ag—it—kin--a—tation" (in a state of agitation). He is very earnest and demonstrative with his hands, which fly all over his body when telling his story of the pit accident. Attention is at once drawn to the feebleness of the labio-dental movement in speaking.

His vocabulary is very limited and his sentences in-

appropriate to his purpose, his words clumsy in their application, not incisive or explicit. This voluminous, almost meaningless, speech is very characteristic. Talks in a monotonous, very confidential tone of voice, also characteristic of many epileptics. The following is an extract from a letter written to "Mr. the Governor Inspector of Scotland": "When I write to the Governor Inspector in 1881 and the answer that I got back on Christmas morning was my dead letter that I was to come out through death into life under her Magast serves and now the time that I have been in I would like you to judge my case in a medum way according to the rules of the Scriptures, and the rules of the laws," etc.

Case 5. A. F., æt. 24, of dark strumous type, with bad family history of strumous character. She is weak-minded and childish, and has had no education.

Her imbecile condition is rather a hindrance to our obtaining a correct conception of the mental side of her speech faculty, for she is weak of understanding, illiterate, and incapable of any subjective study of memory or recollection. Her utterance is slow, thick, and, except when excited, anergic and muttering in character. Certain conjunctions of syllables she is unable to bring out, such as "br" in February—she says "Fetherwary." Her memory is weak. She says she is four months here, whereas she has been four years. Echolalia is at times very marked, *e.g.* she repeats in reply to questions, "a' thegither," "I wish—I wish—my airm—was better a' thegither;—I wish—I was a wee better—a' thegither;—I wish I hadna been here—a' thegither." Addressing the nurse, she says, "My granny stays at Kilmarnock—she's a puir auld woman—my granny—a puir auld woman—I cam—tae stay here—tae bide—afore ma puir auld mither deed—ay, ma puir mither deed—I used—tae wash—ma puir mither's hearth stane—Is your puir mither no deed?" A negative reply. "When are ye—gaun tae see her—wull ye—tell your puir mither—that A. F. was speerin'—for her—wull ye—tell her that puir Agnes—has got a sair airm?" There is bradylalia noticeable as well as echolalia, a limited, very limited, vocabulary, shallow respiration, and feebleness in the oral mechanism.

The strain of the foregoing speech indicates, what is more noticeable in the tone of the voice, the emotional character of the patient. The simple statement—"Is this Monday?—then yesterday—would be—the Sabbath day"; the last words uttered with reverence shows her religious emotionalism—it cannot be called in her case intellectualism.

Imperfect as this case is from the clinical student's point of view, because of her weak intellect, it is in some degree a contribution to the subject of undoubted value.

Case 6. R. B. L., æt. 22. Insane at age of 18. Is rather dull intellectually, religious emotionalism marked, and religious delusions scarcely absent at any time. He takes few seizures of *grand mal* or *petit mal* types. They are more frequently mental and automatic in character, and he has no recollection of them afterwards. The hypochondriacal element is here very prominent. He has a dazed, far away expression, with a tinge of sadness in it, an expression as being "not of this world."

Asked regarding his speech, and what difficulties he noticed, he replied in halting speech and evidently with some mental confusion, "When—I have come—the right way—to a speech—I know very well how to speak to any person." He can repeat verses of psalms and hymns with fair promptitude, but intellectual operations are slow, and he puts in wrong words, so that the meaning is confused. Innervation of vocal and oral mechanisms is fairly good, the speech defect being more mental and amnesic than motor. There is very little muscular tremor, no agraphia, word blindness or word deafness. Asked if he ever took a fit, he replied, "I would count the darkness for the fit," meaning that his sight failing was the first sign, and then he added, "I knew myself—I would do better every day if—I was within the fresh air." His memory is best when talking of religious matters, the text or heads of last Sunday's sermon, etc. He is demonstrative in his speech, points with his hand all the time, says "praised" for praying, "meals meat" for meal of meat.

Case 7. E. F. D. has been subject to fits for years, exact period unknown. She has had several severe illnesses during

the last few years, *e.g.* an attack of coma with high temperature for several days at one time, and acute bed sore at another. She has been much reduced in strength. The majority of her fits come on at night, and if she has a night fit she is usually excited till she has two more. She is amnesic. As I look at her she holds out her right hand, which is trembling, and says, "I—don't—don't," and then there is a long pause, and when I fill in what I think is the rest of her sentence by saying, "you don't feel power in your right hand," she promptly replies, "No, I don't." When I remark, "You seem to have a difficulty in remembering words," she replies, "Weel—I just be—no—kind—o'—," a long pause as if paralysed, and then the end of the sentence is uttered, "the rale thing just." Questioned "Do you sometimes say the wrong word—the word you don't mean to say?" she answers promptly, "I do." There is no word deafness, and if she could read there might probably be evidence that there is no word blindness, though her sight is affected after fits. There is at times distinct echolalia. To the question, "How old were you when you took the first fit?" she replied, "My—my—mither—mither—no—that—I ken o'—I dinna ken o'—I had—to—go—to—work—I had to go to work when I was ten years old—I had—to—I had—to—I had to work that's—just—the—truth. She doesna ken what she's—talkin' aboot—ma mither said—ma mither said—ma mither said—there was ane—o'—thae—kin o'—catch—thae fits—and—." As here indicated, bradylalia is well marked. Asked her age, she answered, "I'm older than thirty years of age now." Speaking immediately after of her husband's pay, she said, "He had mair than thirty years," meaning thirty shillings a week. Her memory generally is impaired. She cannot tell at what hour she gets breakfast, dinner, or tea. When excited, amnesia and aphemia are less noticeable. She is only slightly demonstrative when speaking, except when excited. She talks in her quiet moods in a confidential manner, hesitating very much at times, and in a low voice, the lips and jaws parting slightly and the respiration being very quiet and feeble. Tremors of all the facial muscles are

noticed, and still more so, tremors of the hands, especially the right. She puts her fingers to her lips when trying to speak, as if conscious of muscular inertia, and from a desire to help her utterance. The speech defects in this case, memorial as well as motor, are more marked probably than in any of those previously quoted, though M. B. and P. H. are both very bad. These three are amnesic and dysphasic in a marked degree.

Case 8. D. R., æt. 25, a miner. Has taken fits at varying intervals from the age of eighteen. When asked if he has noticed any difficulty with his speech, he replies, "There is something away from my speaks—and my—memory." There is no agraphia, he understands what is said to him. He reads correctly, but in a somewhat sing-song tone, raising his voice at the end of every sentence, and pronouncing his words in rather a snappish manner. There are no tremors. When shown a sheet of foolscap, and asked to give it a name, answers, "Well—it's—a"—pause—"you can't say it's a book—but—it's a pretty tidy book it—would—do—a grocer," meaning doubtless that it would do for wrapping paper. When shown an envelope, he replies, "That is a tidy—envelope"; shown a watch, answers, "Well—it—will—be—an English—lever." Here there is again the redundancy already noticed, to cover amnesic difficulties, and bradylalia is quite noticeable. There is undoubtedly motor difficulty in this case also.

Case 9. B. C. This patient is an old asylum resident and was regarded as an epileptic twenty-five years ago. She has of late years been much less subject to fits, and has not had one for nearly a year. She is bright, active, and fairly intelligent, considering her long residence in an asylum. She can give a fairly correct account of her own case. When asked, "Have you ever any difficulty with your speech?" she answered, "No this long time. I had when I used to take fits—it was next morning—I couldna speak right. The attendents knew from my speech in the morning when I had taken fits. I couldna get the *full* word out. I knew what I was going to say, but I couldna get the *full* word out." Here there was dysphasia but no amnesia.

Many more cases might be cited in detail, all confirming those which have just been described, and before summing up I will merely give brief statements regarding a few.

Case 10. A male patient illustrates redundancy of speech as if conscious of amnesic defect, by answering the question, "What's this?" (book) thus—"A sort of library book."

Case 11. A male patient illustrates various defects of articulate speech. He has noticed after fits that his speech wanted strength. The emissive energy is spent before the sentence is finished, and it dies away in inaudible words. There is aphonia therefore. The muscular energy is feeble, and the respiratory movements restricted. In his own words he adds, "I have many many times noticed a difficulty in finding words to express myself." There is therefore amnesia also.

Case 12. A male patient has thick, hesitating, drawling speech.

Case 13. A male patient says he is an elegant speller, and is confused because he has not used the right word which should be "excellent."

Case 14. A female patient says she and her brother were both stutterers when they were young. Her sentences are broken, and there is a circumlocution in describing events and circumstances. Her memory for words fails at times, especially after fits, and when trying to speak she feels as if her tongue were paralysed.

Case 15. A male patient describes his speech defect thus: "I feel a little now—not able to come to the point—have the word ready, but I can't get it out—tongue would not come forward at the proper moment." After fits he is always at a loss for words.

CONCLUSION.

To sum up, I think it will be generally accepted, that a considerable range of speech disturbance is to be found in epilepsy, that there is much resemblance in the cases, and yet individual diversities, and that when the normal mental habit is resumed

after one of those periodic outbursts of motor-mental excitement which characterize epileptic insanity, the power of speech is diminished. It is established (1) that before and after fits amnesia and dysphasia are marked, (2) that when there is emotional excitement these conditions are altered according to the degree of the emotional excitement, (3) that when the ordinary mental habit is resumed, and nervous tension has disappeared, the patient suffers from reaction, which tells on his speech faculty by reducing the energy of the memorial and motor centres.

The Patient's Consciousness of Speech Defect. Although, as already observed, they incline at first to denial, they usually admit it when their own stumblings find them out. Their facial expression is quite sufficient to demonstrate that they are anxious and disturbed when their speech is being tested, and there is manifest effort, in the halting yet deliberate speech, which reveals that the patient is anxious not to make mistakes. This is seen also in the careful answers to such questions as, "What is this?" (a key *e.g.*), answers characterized by apologetic introductions, or qualified by unnecessary adjectives. The tremors are often worse, and the break in the voice worse, when consciousness of a difficulty renders the patient emotional.

Emotions as Affecting Speech. This is true of most people, but emotional speech is rarely excited in ordinary circumstances, just because emotions are not so acute, and are more under control. Just as we may have hysterical aphonia, the result of emotional disturbance, so there may be in the epileptic respiratory spasm from a like cause. Undoubtedly the emotional element must be taken into account in considering the different speech abilities of the epileptic at different times. While this applies to human speech generally, it applies in a marked degree to the epileptic. The pantomime of the epileptic is sometimes vivid, frequently profuse and redundant like his speech, and indicates the emotional nature of the man.

Amnesia. It will not be considered beyond a few words how much this is due merely to the defect of recollection, but

it may be said in passing, that as the amnesic state is at times more marked than at others, the *retentum* may be in memory, though not always forthcoming. It depends on special sensations, and the particular emotional state, happiness, anger, rage, etc., and on the degree, whether the faculty of recollection is stimulated or inhibited. I have already pointed out that the range of vocabulary is more or less limited with most epileptics, and this is probably due to failure of memory (loss of retention) apart from failure of reproduction (recollection).

Aphemia and Dysphasia. Extreme aphemia is rarely observed, and then only for a time, usually before and after fits. Dysphasia best describes the articulate speech of the epileptic. Here we have to take account of the vocal mechanism, taking along with it the respiratory mechanism. It may be taken, speaking generally, that there is usually a reduction of emissive energy of all these mechanisms from faulty innervation, and that there is want of synchronous coordination. Hence we may have feeble, stuttering, or staccato speech, and weak or spasmodic glosso-labio-dental movements. We have also sensations of "a catch in the breath," loss of phonation, or reduction of it, as seen in the growing weakness of voice at the end of a sentence. This points to nervous spasm or reduced innervation of the vocal and respiratory mechanisms. Tremors of the facial muscles, of the labial in particular, and tremor of voice indicate unstable innervation.

I need only mention in a few words *Bradylalia*, which has been abundantly demonstrated, and *Echolalia*, which is less common, but sufficiently frequent to call for notice here. *Agraphia* has not been noticed, but those patients who could write were asked to sign their names, and there was found a tremor, sometimes continuous, but mostly interrupted, in their writing, suggestive of alcoholism.

CASES FROM THE WARD FOR DISEASES OF WOMEN, GLASGOW ROYAL INFIRMARY.

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CASE 1.

Haematoma of left tube and ovary occurring during pregnancy. Left hydrosalpinx. Removal by abdominal section. Pregnancy un-interrupted.

MRS. C., aet. 27. One child eighteen months ago. Admitted 10th February, 1899.

Report on Admission.—Patient has not menstruated for three months, the last period occurring in end of October. For the last two and a half months she has not felt well, being weak and easily tired. She also was troubled with nausea in the morning though she never vomited. She never had any discharge, nor was there any abdominal pain till the present attack.

On Tuesday, 7th February, she complained of a sharp cutting pain in left iliac region, so severe as to cause her to cry out and to constantly change her position in bed in attempts to get relief. She had no discharge of blood. This pain has continued, with occasional extreme paroxysms, without much intermission up to the present time, and is increasing in severity. Her diet has consisted mainly of milk and soda-water, as solids increase the severity of the pain complained of. She took large doses of purgatives with the intent of getting relief, but without avail. The bowels moved freely on the 7th, but have not moved since. On the 7th and 8th

the pain was so severe as to prevent her sleeping. On the 9th she slept after a morphia suppository. She has not had any rigors nor any collapse, but she states that on Sunday, 5th February, when in church, she almost fainted, but the faintness was not preceded or accompanied by any abdominal pain. Patient was quite well after her confinement eighteen months ago, which was easy and uncomplicated, and nursed her child for ten months. In her first pregnancy she had no morning sickness, but this has been marked during the present pregnancy.

Patient is flushed, and has an expression of being in pain. Pulse 106, resp. 26, temp. 99.6° , rising at night to 100° . A hypodermic of morphia given, $\frac{1}{3}$ gr., and at midnight again $\frac{1}{4}$ gr.

11th February. *Physical Examination*.—Urine normal. Cardiac and pulmonary signs normal. Breasts secreting. Abdomen is rather protuberant, dulness reaching above umbilicus, but on removal of 22 oz. of urine, dulness and feeling of tumour come about two inches below umbilicus. Hypogastrium is extremely sensitive to pressure, tumour is slightly movable from side to side, but manipulation causes great pain. Per vaginam the cervix is found enlarged and softened, close behind symphysis. The posterior part of pelvis is occupied by an irregular, tense, and extremely sensitive tumour, which presses into the rectal lumen, presenting there a more cystic feeling. Pain continues and is partially relieved by morphia hypodermically.

12th February. Catheter was again required this morning. Pain, as before, relieved by morphia. Temperature normal.

It was evident that we had here some serious complication of pregnancy, and the diagnosis at which we arrived was ectopic gestation with partial rupture and formation of haematocoele. The faintness on Sunday was taken to indicate an internal haemorrhage, and the distress setting in on Tuesday and continuing till admission on Friday seemed due to intrapelvic pressure and localized peritonitis. Some doubt was raised by (a) the mobility of the tumour in the abdomen, which resembled a pregnant uterus between the fourth and

fifth months; (b) the absence on admission of signs of anaemia such as ought to have followed an effusion of blood sufficient in quantity to cause the tumour felt in Douglas' pouch; and (c) the absence of uterine haemorrhage. But it was possible that there was both an intra- and an extra-uterine pregnancy, and the bleeding into Douglas' pouch might not have been very free, the chief mass of the tumour felt in the abdomen being evidently not haematocele, and possibly unruptured sac. Nothing that we gathered from the history of the patient suggested the condition found at operation.

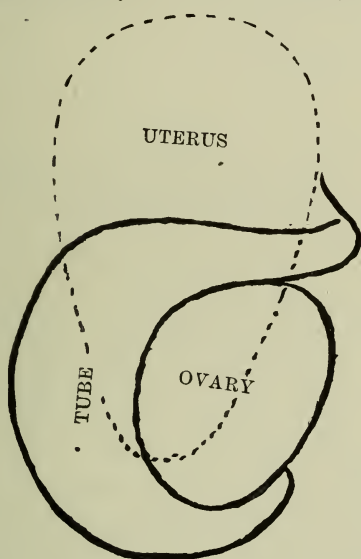
13th February. Under anaesthesia with A.C.E., abdominal section. Uterus of about size of a four-months' pregnancy was found immediately under the parietes, and on tracing down the left tube it was found to be acutely bent backwards and inwards, and adherent to bowel at the place of bending. The uterus was brought out of the abdominal wound, and the ovary and tube, which were black and gangrenous-looking and adherent all round in Douglas' pouch, were shelled out of their adhesions, raised and tied off at the bend. There was hardly a drop of blood lost. The right ovary and tube were normal. The uterus was replaced in the abdomen and the wound closed with sutures passing through the whole thickness of the abdominal wall.

On laying open the dilated tube it was found to contain about 6 oz. of clear, slightly brown-coloured fluid. The wall of tube about $\frac{1}{4}$ inch thick on an average. The ovary, about size of small orange, was completely infiltrated with blood, and the section resembled that of a spleen (haematoma of ovary). The accompanying diagram represents the position of parts, the uterus being in front.

The after-history need not be given in detail. The pain ceased after the operation. The catheter was required three times. The bowels were moved on the 15th. The sutures were removed from the abdominal wound on the 25th, and on the 29th patient was allowed to go home.—*Note.* Mrs. C. was delivered of a female child at term on 6th August, 1899. The labour was natural and easy.

So far as I have been able to trace I find no record of any

case the same as this. Cases of torsion of the pedicle of an ovarian cyst, and even of the normal ovary, cases also of torsion of the tube have been reported, but in this case there was no torsion, but simply an acute bend at the inner ends of tube and ovarian ligament. The condition was undoubtedly due to an obstruction to the flow of blood through the ovarian vessels on the left side, an obstruction also commencing in the veins, and probably therefore due to something of the nature of a twist or kink. The history indicated that this obstruction took place rather suddenly on 5th February. And it is this



suddenness that gives rise to the chief difficulty in the explanation. For a large hydrosalpinx had no doubt been present for some time, probably from a period long antecedent to the pregnancy, and had it been adherent in Douglas' pouch, a gradual impaction in the pelvis might have resulted, and would have caused corresponding symptoms. But while, of course, the tube may have been long located in Douglas' pouch, the adhesions found at the operation could only have been present for a few days, and had evidently originated with the haematoma. Previous to this attack therefore both tube and ovary had been free of adhesions. Something else than

adhesion must have held them down in the hollow of the sacrum while the uterus in its ascent was attempting to pull them up. This something will always remain doubtful, but probably the adhesion to bowel found at the bend was a factor in the cause. By this probably the pedicle of tube and ovary was fixed. The rising uterus dragged up the part of tube and ovarian ligament above the band of adhesion, and made the angle of flexion always more acute until a point was reached at which venous stasis below it commenced. This would at once, by swelling of the parts below, increase the flexion, and from that point the haematoma and necrosis would rapidly proceed.

RECENT CASES OF EXTRAUTERINE PREGNANCY PRESENTING NOTEWORTHY FEATURES.

CASE 2.

*Rupture of early extrauterine pregnancy. Complete disappearance of
haematocoele by absorption.*

Mrs. W., aet. 29. III para. Last child 2 years ago. Admitted 29th March, 1899.

Report on Admission.—Patient was healthy up till three weeks ago, when she had a miscarriage at two months, the last menstruation having occurred on 6th January. At the time of miscarriage the haemorrhage was profuse, and continued to a less extent for eight days. The discharge contained clots at first, but latterly was fluid blood. For the last few days it was serous in character and entirely free from blood. Defaecation is accompanied by pain in the lower abdomen. Micturition is painful and slow, and on one occasion, 24th March, the catheter had to be used. Patient has never had any attacks of faintness, and for the first week continued going about her household work. For the last fortnight she has been confined to bed.

Patient is anaemic. Temp. 101·8°, pulse 80.

30th March. *Physical Examination*.—Urine normal. Cardiac and pulmonary signs normal. The abdomen is rather full in the umbilical region, where slightly distended coils of bowel are evident through the parietes. On palpation a hard mass is felt, forming a ridge passing across lower abdomen from a point three inches to the right of the middle line to an inch and a half to the left, the highest point being at its right extremity, where it is at the level of the A.S. spine. From this ridge on deep palpation the mass is felt to extend in all directions. Per vaginam: the cervix is found displaced forwards; the posterior part of the pelvis is occupied by an irregular somewhat round firm mass continuous with that felt per abdomen. The uterus measures $3\frac{1}{4}$ in. The whole mass is slightly movable. Through the anterior abdominal wall the fundus uteri can be definitely marked off from the tumour-mass lying behind. To be treated by rest, hot moist abdominal compresses and hot vaginal douches.

April 1st. In addition to free movement of the bowel there is a pretty constant discharge per anum of clear jelly-looking semi-fluid matter accompanied by considerable pain and feeling of pressure.

April 4th. Tumour in abdomen diminishing in size, but condition per vaginam very little changed.

April 8th. Very little of the tumour can now be felt per abdomen. The fundus and indeed the whole body of the uterus is now freely movable, but on deep pressure the remains of the haematocele can be felt behind the right of the uterus. Per vaginam also the swelling is greatly diminished, and presents now the form of a very hard and slightly irregular ridge passing transversely across Douglas' pouch.

April 13th Effusion still diminishing.

April 15th. Patient went home to-day. Uterus freely movable; hardly any thickening to be felt behind or to either side.

CASE 3.

Rupture of extrauterine pregnancy at fourth month. Uterine haemorrhage. Abdominal section ten weeks after. Successful removal of foetus, placenta, and tube.

Mrs. M., aet. 38. VIII para. Last eight months ago. Admitted 12th April, 1899.

Report on Admission.—Patient dates the onset of her illness from the birth of her last child, eight months ago, when, just before the birth a severe flooding set in, which went on for six weeks after the birth of the child, accompanied by clots. For a month after that she had no discharge, and then a normal menstrual period occurred. For four months thereafter she had amenorrhoea. This was followed by another flooding, which lasted six weeks, during which she had to keep bed. This flooding came on with cramp-like pains and patient fainted at the time. Nothing like an ovum was observed in the discharge. The discharge ceased for a week and then began again a fortnight ago, being present up till the day before admission.

Since the beginning of February, when this flooding came on, patient has had a severe pain in the right iliac region which has been constantly present, and has been accompanied by rigors and profuse sweatings. For the last six weeks morning sickness has been present. Occasional pains affect the right breast. Is troubled with frequent micturition and sometimes retention of urine. Since the birth of her 6th child, four years ago, she has had occasional prolapse of the womb, sometimes complete.

Patient has an anxious expression, sallow complexion, and is anaemic. Temp. 96·8°, rising in evening to 99·2°, pulse 70.

13th April. *Physical Examination.*—Urine 1030. Trace of albumen. Pulmonary signs normal. Blowing V.S. murmur. Areolae dark, some secretion from left nipple. Abdomen large and flabby, flattened rather than protuberant. On palpation a firm resistant mass is felt above the pubis reaching to about two fingers' breadths below the umbilicus and passing more to

right than to left of middle line. Tenderness is more marked on the right side. On deep palpation this mass seems to extend in all directions. Percussion is not absolutely dull except over a small region above the symphysis and slightly to the right.

Per vaginam: the vaginal walls are very soft; cervix enlarged and softened, presenting enlarged follicles; right side of pelvis and Douglas' pouch are occupied by an irregular firm mass; uterus lies close behind pubis, cervix is in usual situation. Uterus measures $3\frac{1}{2}$ in., fundus lying directed slightly to left. No haemorrhage in the meantime.

For the next three days patient continued in much the same condition, and on 16th April there was a return of the uterine haemorrhage. Accordingly:

17th April. Under chloroform. Abdominal section. Right tube was found distended in outer part to about a diameter of two inches with blood-clot. Below this a four months' foetus was found of a slaty-green colour and with tissues softened. Outer end of tube was torn off from the rest during removal and on raising it up separately there was some bleeding from the ovarian artery. This was easily controlled by ligature and suture, and there was no other haemorrhage. The placenta was removed along with the enlarged tube to which it was partially attached. Douglas' pouch was cleared of several clots. The abdominal wound was closed with a single line of sutures.

The further course was uneventful. The sutures were removed from the abdominal wound on 29th April, and on 5th May patient left the hospital.

These two cases, which were equally happy in their ending, present instructive features both of resemblance and of contrast which deserve a brief note.

They both occurred in multiparae; they were both right-sided in origin; in both there was profuse uterine haemorrhage suggesting abortion, and in both an abdominal tumour resulted.

The points of contrast may be more distinctly brought out if arranged in a tabular form.

A.

B.

(a) Stage of advancement of pregnancy at time of rupture.

Early, probably not more than a month. About four months.

(b) Phenomena accompanying rupture.

No sudden faintness. Sudden faintness.
Gradual loss of strength. Immediate loss of strength.

(c) Sequelae of rupture.

No urgent uterine symptoms. Continued and alarming uterine
Uterine haemorrhage gradually haemorrhage.
 ceasing. Inflammatory reaction (rigors,
No inflammatory reaction. sweatings, probably elevated
 temperature).

(d) Abdomino-pelvic tumour.

Gradual rapid disappearance. Persistence.

(e) Pain.

Gradual and finally complete Continuous with frequent exacerba-
cessation. tions.

It is impossible to lay down absolute rules for the treatment of all cases of extrauterine pregnancy. But while in view of the immediate danger from haemorrhage, which is to be dreaded even in the earliest stages of pregnancy, it ought to be a rule to operate on all cases at once on the occurrence of rupture, this rule does not apply when the patient has survived the rupture and its consequent haemorrhage. The treatment must then be based on the expectation we can form as to the course that is likely to be taken by the haematocele and its contents.

In my opinion the two cases detailed above illustrate well the principles that should guide us in this expectation. In A, seen for the first time three weeks after rupture, the symptoms gradually diminished in urgency, the tumour was rapidly absorbed, and the uterine reaction gradually ceased. The embryo had died at probably so early an age that its absorption would not be more difficult than the absorption of ordinary blood-clot. In B, on the other hand, the pregnancy had advanced so far before rupture took place that the foetus and placenta would form materials exceedingly difficult of removal

by absorption, if indeed such removal could at all be considered possible. The rupture had taken place ten weeks before the patient was first seen, and there was still a considerable abdomino-pelvic tumour. Absorption might have, and probably had, occurred to some extent, but had stopped far short of complete removal of the haematocele and its contents. Besides this there was severe and long-continued uterine haemorrhage, which was itself an evidence of the malign influence of the diseased pelvic condition. To this I have referred in a paper on Extrauterine Pregnancy in the *Glasgow Medical Journal* for June, 1898, and I regard it as an important indication of the necessity for operative interference. To all this was added the fact that the whole organism was affected injuriously by the pelvic inflammation constantly present and frequently aggravated.

Probably therefore it should be regarded as a trustworthy rule in the cases seen some time after rupture has occurred, not to interfere except by assisting absorption when the tumour is lessening in size and uterine reaction is absent, but in all other circumstances to operate.

It will be observed that in Case B the patient came under observation eight months after she was delivered of her eighth child. Two-and-a-half months after delivery she had what she considered a normal menstruation, and this was followed by the extrauterine pregnancy which was terminated by rupture at the end of four months. There was therefore an interval of only two and a half months between the intra-uterine and the extrauterine pregnancies. A still shorter interval—about four or five weeks—seems to have marked the following case, which is remarkable in other respects.

CASE 4.

Rupture of extrauterine pregnancy at second month. Uterine haemorrhage. Abdominal section six weeks after. Removal of tubal mole. Recovery complicated by uraemic (?) symptoms.

Mrs. Q., aet. 21. One child four months ago. Admitted 11th May, 1899.

Report on Admission.—Patient complains of severe pain in the lower part of the abdomen, accompanied by haemorrhage, which has been present for about six weeks. Four months ago she had a child, which she nursed for two months. Menstruation returned a month after the confinement, appearing normal in character. This was followed by a period of amenorrhoea for eight weeks, but without morning sickness. This was succeeded by a pain in the left mammary region, which patient thinks was due to a fall. This pain was so severe that it affected her breathing. It has gradually moved down to the left iliac and hypogastric regions, and has been present ever since, and has been so severe as to prevent her sleeping at night. Three days after the onset of the pain discharge from the uterus came on, and has been present since, although it has not been much in quantity. At first there were stringy materials in it. Dyspareunia has been severe since the onset of the above symptoms. Defaecation has been painful for the last fortnight, but no blood has passed per rectum. The faeces were dark in colour. Patient has not fainted at any time during the present illness. She has always been a healthy woman.

12th May. *Physical Examination.*—Patient is florid and well developed. There is a feverish flush on cheeks. Tongue coated with white fur. Temp. 101.2° , pulse 105. Urine albuminous. Cardiac and pulmonary signs normal. Respirations 20. Abdomen distended. Tenderness on palpation over iliac and hypogastric regions. Fulness in left iliac region passing across into right side. Dulness with resistance over the whole tender area. Per vaginam: uterus is carried forward near, but not quite close to symphysis. Whole posterior part of pelvis is occupied by a tense and rather elastic tumour continuous with that felt per abdomen. There is some uterine haemorrhage.

The pain and haemorrhage continued after admission, and the general condition deteriorated. Temperature rose on 13th May to 102.2° . The pain was exceedingly severe, and necessitated the use of morphia hypodermically every evening, and even with that the sleeplessness persisted.

14th May. *Abdominal Section*.—Omentum greatly thickened and blood-stained, adherent to abdominal parietes and to sac of haematocele. Bowel also adherent over haematocele. Sac opened and cleared of clots. Right tube and ovary removed. Tube in nearly its whole length about an inch in diameter and filled with firm blood-clot, which slightly protruded at the gaping ostium abdominale. No embryo found. Glass drainage tube left in.

So far as regards the extrauterine gestation, the further history of the case might be summed up by simply saying that the recovery was uninterrupted. The drainage tube was removed on 17th May. The gauze drain then left in was removed in other twenty-four hours. The bowels moved on the day following the operation. The urine was passed naturally. The sutures were removed on 28th May, and patient left hospital on 6th June.

But it will be noted that the patient had albuminous urine, and that she several times had morphia injections for the relief of the pain, and it is possible that these two facts account for the phenomena that followed the operation, and that were at first suspected to indicate a commencing septic meningitis. The report is so interesting that it is worth giving in full for the first few days after the operation.

14th May. Operation at 12 noon.

12.30 p.m. Temp. 98·6°, Pulse 94, Respirations 31. Had a stimulant enema.

1 p.m. Very restless. Morph. hypod. $\frac{1}{3}$ gr.

4 p.m. T. 100·6°, P. 106, R. 24. Nutrient enema. Dressings stained; changed.

8 p.m. T. 103·2°, P. 95, R. 22. Passed 11 oz. urine naturally. Nutrient enema with 20 gr. quinine.

12 p.m. T. 102·2°, P. 100, R. 16. Had morphia $\frac{1}{3}$ gr. hypodermically at 10 p.m., and was sponged with spirit and water. Has been sleeping for a short time. Passed 3 oz. urine. Had 4 oz. nutrient enema.

15th May. 4 a.m. T. 102°, P. 110, R. 16. Has been quiet, dozing nearly all the time. No sickness. Passed 6 oz. urine and flatus from bowel.

- 8 a.m. T. 101·8°, P. 112, R. 14. Bowels have moved freely. Urine lost.
- 12 noon. T. 102·4°, P. 112, R. 20. Has had sips of hot water, 6 drachms brandy and water and 4 oz. beef tea by the mouth. Passed 8 oz. urine. Dressings stained; changed.
- 4 p.m. T. 102·4°, P. 108, R. 17. Has had $\frac{1}{2}$ oz. brandy and 9 oz. soda water and milk. Passed 4 oz. urine and a small hard faecal motion. Has dozed a good deal.
- 8 p.m. T. 102·4°, P. 112, R. 18. Had a seidlitz powder and has had several loose stools; seems to have defective control. Has had 3 oz. soda water and milk and $\frac{1}{2}$ oz. brandy.
- 12 p.m. T. 102·8°, P. 120, R. 16. Has had 12 oz. milk and lime water, $\frac{1}{2}$ oz. brandy, and $\frac{1}{3}$ gr. morph. hypod. Passed 5 oz. urine.
- 16th May. 4 a.m. T. 102·6°, P. 120, R. 18. Has had $\frac{1}{2}$ oz. brandy, 6 oz. milk and lime water. Vomited a little watery fluid. Has had one small loose stool.
- 8 a.m. T. 101·6°, P. 116, R. 16. Has passed 8 oz. urine. Has had 16 oz. milk and lime water and $\frac{1}{2}$ oz. brandy.
- 12 noon. T. 101·4°, P. 114, R. 16. Has had 8 oz. milk and lime water. Passed 3 oz. urine. Dressings changed.
- 4 p.m. T. 101·4°, P. 115, R. 17. Has passed 3 oz. urine. Vomited twice. Has had 8 oz. milk and soda water.
- 8 p.m. T. 100·4°, P. 116, R. 16. Has passed 6 oz. urine. Had 1 oz. brandy and 1 oz. milk and soda water.
- 12 p.m. T. 99·4°, P. 114, R. 20. Has vomited a good deal. Slept and dozed a good deal, but for a short time was very excitable. Had 2 drachms brandy with soda water and 4 oz. nutrient enema.
- 17th May. 4 a.m. T. 99°, P. 108, R. 20. Has passed 9 oz. urine. Had $\frac{1}{2}$ oz. brandy and 6 oz. milk and lime water. Vomited sour smelling brown fluid.
- 8 a.m. T. 98·6°, P. 106, R. 20. Has had 4 oz. nutrient enema. Has been at times very excitable. Has vomited twice. Has been very unreasonable, and insists on moving about in bed. Had a free movement of bowels and passed urine. $\frac{1}{3}$ gr. morph. hypod.

- 12 noon. T. 100° , P. 121, R. 11. Has been very noisy and obstreperous, sometimes lies almost on her face. Face livid. Teeth clenched. Pupils widely dilated. Passed urine in bed. General twitching of face and hands after the outburst. Has had 3 oz. nutrient enema.
- 3.30 p.m. T. 101.6° , P. 124, R. 12. Has had 8 oz. nutrient enema. Passed 6 oz. urine. Patient has dull lethargic expression. Face flushed and breathing Cheyne-Stokes in character. Pulse full and bounding. Has lain all day without speaking or appearing to know what is going on around her; sometimes giving vent to piercing screams when she is touched. Pupils contracted and equal. Vomits fluids given by the mouth. Passes urine and faeces in bed. Hair is cut off. Ice bag applied to head. Blistering fluid to nape of neck. Dressings changed. Drainage tube removed.
- 8 p.m. T. 101.6° , P. 114, R. 13. Had 4 oz. nutrient enema. 4 oz. urine removed by catheter. Vomited some brown fluid. Had hypod. of strychn. ($\frac{1}{80}$ gr.) and digitaline ($\frac{1}{100}$ gr.) at 5 p.m.
- 12 p.m. T. 99.6° , P. 122, R. 15. Swallowed 2 oz. milk in sips. Vomited 3 oz. odourless brown fluid. Screamed twice, but was not so tremulous as before.
- 18th May. 4 a.m. T. 98.4° , P. 120, R. 20. Has had 8 oz. nutrient enema. Swallowed 2 oz. milk. Had another hypod. of strychn. and digital. Has passed urine and faeces in bed. Screamed once and cried as if frightened.
- 8 a.m. T. 100.6° , P. 120, R. 20. Has sipped 1 oz. milk. Had 4 oz. nutrient enema. Has cried several times. Passed urine and faeces in bed.
- 12 noon. T. 100.8° , P. 116, R. 17. Had 4 oz. nutrient enema. Hypod. strychn. and digital. Catheter has been kept in bladder, and 14 drachms urine caught in receiver. Has been moving both arms and legs, and swallowed a little milk now and again.
- 4 p.m. T. 100.2° , P. 116, R. 23. Dressings changed. Is more sensitive, but still in a stupid condition. Conjunctival reflex present. Pupils widely dilated. Breathing regular. Occasional vomiting of yellow-coloured fluid. Passed $4\frac{1}{2}$ oz. urine.

8 p.m. T. $100\cdot2^{\circ}$, P. 103, R. 22. Had $\frac{1}{8}$ gr. pilocarp. nit. at 4 p.m. Has taken 4 oz. milk. Had 4 oz. nutrient enema. Skin now moist, but no free perspiration. Passed $4\frac{1}{2}$ oz. urine.

12 p.m. T. $100\cdot2^{\circ}$, P. 112, R. 26. Had again $\frac{1}{8}$ gr. pilocarp. and strychn. and digital. Has taken 5 oz. milk. Passed $3\frac{1}{2}$ oz. urine. Has vomited very little. Has been slightly restless, and has tried to speak.

19th May. 4 a.m. T. $99\cdot8^{\circ}$, P. 110, R. 26. Has been at times very restless, tossing from side to side. Is now sensible, and answers when spoken to. Has had 7 oz. milk.

The crisis was now past, and by the afternoon of the 19th the intelligence was completely restored, appetite and the power to swallow returned, and there is nothing further to note in the future course, except an eruption of herpes on the lower lip on the morning of 21st May.

There can be little doubt that the phenomena in this case were due to uraemia, and in that view the following points are perhaps worthy of special note:

(1) The urine was rather scanty all the time. It was always albuminous, but no tube casts were detected, and the albumen was never in large amount. The scantiness of urine was most marked, and indeed amounted to almost complete suppression in the twelve hours immediately preceding the marked slowness of respiration and the period of profound stupor on 17th May.

(2) The first marked slowing of respiration on the morning of 15th May followed the administration of morphia. The next hypoderm of morphia was succeeded twenty-four hours by respiration varying from sixteen to eighteen per minute. The last morphia hypoderm preceded by only a short interval the most marked slowing of respiration on 17th May.

(3) Scantiness of urine, slowing of respiration, and stupor, seemed to coincide in time and vary together.

(4) The variations of temperature and pulse did not correspond with those of the urine, respiration, and stupor.

(5) Improvement set in with the stoppage of morphia, the

administration of strychnine and digitalis, and the use of counter-irritation to the head.

(6) Improvement was hastened (by?) after the use of pilocarpine.

CASE 5.

Rupture of extrauterine pregnancy at fourth month. Severe pain and haemorrhage for seven weeks. Abdominal section. Removal of right tube, placenta, and foetus. Rapid recovery.

Mrs. M'T., aet. 24. Ipara, nine years ago. Admitted 31st May, 1899.

Report on Admission.—Seven weeks ago patient was suddenly seized with a severe pain in the right iliac region, which has been present more or less ever since. She did not faint at the onset of the pain, which came on when she was quietly standing in her room, but she felt sick and unfit for any exertion. She has had to stay in bed ever since on account of the pain and weakness. After the pain came on she had a reddish vaginal discharge, which came on almost immediately, and has been present continuously ever since until last week, when it entirely ceased. She has had no rigors nor sweatings, nor pain in the mammae.

She was treated by her doctor for a miscarriage, but as she did not improve she was sent from Islay to the Western Infirmary, where she remained about three weeks. During this time she was twice under chloroform, but her case was considered a doubtful one, and she was sent back to her home in Islay three weeks ago. After her return the pain and discharge were very severe. She vomited a great deal, and was unable to sleep. Her cries were so distressing that the whole neighbourhood was alarmed.

In the vaginal discharge there have frequently been fleshy-like masses, like blood-clot, and sometimes even like skin. Patient thinks the abdomen has slightly increased in size since she was in the Western Infirmary.

Menstruation had always been regular up till four months before the onset of the pain, when it ceased. Patient at this time noticed the abdomen becoming larger, but had no morning sickness, nor did she think that she was pregnant.

Physical Examination.—Patient is thin and pale, and has a worn, exhausted expression. Tongue coated with a white fur. Temp. 101°. Both mammae secreting. Lungs healthy. A.S. cardiac murmur with accentuation of second sound. Abdomen large, with thick walls somewhat prominent, especially above umbilicus. Firm mass felt occupying centre and left side of abdomen up to two finger-breadths below umbilicus, somewhat irregular on surface. One part especially dense lies midway between symphysis and umbilicus, more to left than right of middle line. Percussion is clear over this down to about 2 inches above symphysis. P.V. cervix is close behind symphysis. Posteriorly to it the pelvis is completely filled by a firm, slightly doughy mass presenting an irregularly rounded surface. Sound passes in ordinary direction $2\frac{1}{2}$ inches. Flexible bougie introduced passes 4 inches, and causes a slightly sick feeling. Very little tenderness.

There was no doubt about the diagnosis of this case. The only thing calculated to throw doubt upon it was the fact that the patient had been so recently examined by a specialist in gynaecology, who had first regarded the case as one of retroverted pregnant uterus, and then as possibly some cystic condition. These are familiar mistakes in the diagnosis of extrauterine pregnancy, and the fact that they had been made indicated that the haematocele had been present in this case all the time.

After admission the patient continued in great distress, and only found relief from frequently repeated hypodermics of morphia. On 3rd June the tumour was, if anything, larger, and we feared some fresh haemorrhage might be occurring. Accordingly, 3rd June, under chloroform, abdominal section. Large mass of blood-clot and placental tissue connected with right tube removed. Partially macerated foetus enclosed in membranes, evidently about the age of three months, was found

in the centre of the mass. There was almost no haemorrhage, and after washing out the cavity a glass drainage tube was left in.

Patient was at once relieved of her suffering, and next morning had nothing to complain of except want of food! The drainage tube was removed on 5th June. Sutures were removed on 14th June, and patient went home on 22nd June.

It is a curious fact that immediately after her return to Islay, another case of extrauterine pregnancy was sent to us from that island.

CASE 6.

Case of early right-sided tubal pregnancy, ruptured two months before admission. Continuous pain and haemorrhage. Abdominal section. Removal of both right and left adnexa. Uninterrupted recovery.

Mrs. M'Q., aet. 31. VI para, last a year ago. Admitted 1st July, 1899.

Report on Admission.—Patient has a dull heavy expression—is well developed—tongue clean—teeth good. Complains of pain in the right iliac region, accompanied by haemorrhage. This discharge came on about two months ago, and has been present more or less ever since. The pain did not come on until a few days after the discharge appeared. Patient had her last child a year ago. It died a fortnight after birth, owing to “pains and weakness.” Since that time she has never felt quite so well as formerly, although there was nothing definitely wrong. Since the onset of the discharge she has felt weak, but has been able to go about her work.

Menstruation had been perfectly regular, the discharge coming on about the time menstruation was due. Defaecation has been painful, and for the last few days she has passed mucus by the rectum, but no blood. When the pain was very severe patient felt sick, but did not faint.

Physical Examination.—Urine sp. gr. 1030, acid. Trace of albumen. Pulmonary signs normal. Cardiac A.S. murmur.

Abdomen has lax flabby walls, somewhat protuberant in lower part, especially above the pubis, where on palpation a firm mass is felt, presenting a rounded prominence situated a little to the right of the middle line, continuous with a firm somewhat irregular mass passing backwards towards the promontory and leftwards towards the A.S. spine. The rounded prominence above mentioned can be moved in a lateral direction without causing pain. The greatest pain is caused by pressing along the right border of the mass. P.V. cervix is immediately behind the pubis, being pressed forward by a very firm uneven mass filling Douglas' pouch and passing across on both sides to the wall of the pelvis. Very little movement can be communicated to it bimanually. Palpation is much less painful than per abdomen. Sound passes $2\frac{1}{2}$ in., entering the rounded prominence above mentioned lying behind pubis.

At operation on 7th July the pregnancy was found connected with the right tube. This with the haematocele was removed. Right tube was also removed, being found closed and cystic. Glass drainage tube was left in Douglas' pouch.

Recovery was unmarked by any accident. From the day of operation patient was completely relieved. The drainage tube was removed next day, the sutures on 17th July, and the patient went home on 25th.

It is a striking illustration of the frequency of this disease to find two cases occurring in one little western island, and with less than a month's interval between them.

SOME RECENT NOTEWORTHY CASES OF MYOMA.

CASE 7.

Postclimacteric submucous myoma protruding at vulva; gangrenous; removed per vaginam by morcellement.

Mrs. C., aet. 48. V para, last twenty years ago. Admitted 7th April, 1899.

Report on Admission.—Patient has a yellow tinged complexion with marked anaemia of skin and mucous membranes, is flabby and exhausted looking. She states that about five years ago she felt a bearing down pain in abdomen accompanied by a swelling to the left of the hypogastrium. The swelling gradually increased in size, and was associated with severe floodings at the menstrual periods. For about three years before this she had noticed that menstruation occurred every fortnight, with an increase in the quantity of the flow. She was sent into the Western Infirmary in March, 1895, but was only treated with medicines, as operation was considered inadvisable. At first when she went into the Infirmary she had an attack of haemorrhage, but this did not return for some time after leaving, and was only present to a slight degree, being caused by any undue exertion. Menstruation ceased about a year ago, and she felt fairly well till about six months ago, when she noticed the abdominal swelling increasing in size. During the last year she has noticed a slight stain on her linen, but no haemorrhage has been present. On the day of admission she had a discharge resembling menstruation. Micturition is frequent and accompanied by a burning pain during the flow; the quantity of urine is very small.

Physical Examination.—Nothing abnormal in thorax. There is a large tumour in the umbilical and hypogastric regions chiefly marked to the left of the middle line. It is solid in consistency, not tender to palpation, and somewhat movable from side to side. P.V., there is a large pendulous irregular mass protruding from the vulva, sloughing on the surface and giving a very disagreeable odour.

As patient was evidently suffering from septic absorption, the sloughing surface was at once removed by curette and scissors, and iodoform dressing applied. Next day a large portion of the tumour filling the vagina was removed by morcellement, and the iodoform packing renewed. From the size of the tumour I at first thought that it would be impossible to remove it per vaginam, and resolved on an abdominal hysterectomy. In view, however, of the risk of peritoneal infection from the necrotic mass, I resolved to make

another attempt per vaginam, and on 24th April was fortunate enough to get the whole tumour away piecemeal. It proved to be a purely submucous tumour, springing mainly from the posterior wall of the uterus, and after I had removed about three-fourths in pieces varying, say, from a plum to a turkey's egg in size, the remainder of the tumour easily shelled out of its capsule. There was very little bleeding, and the wall of the uterus was unwounded. The cavity left was packed with iodoform gauze. Patient was out of bed six days after, and went to the Convalescent Home on 5th May. She has rapidly and to a remarkable degree regained colour and strength. The whole weight of the tumour removed was about $3\frac{1}{2}$ lbs.

CASE 8.

Myomata complicated with ovarian cyst. Removed by abdominal section.

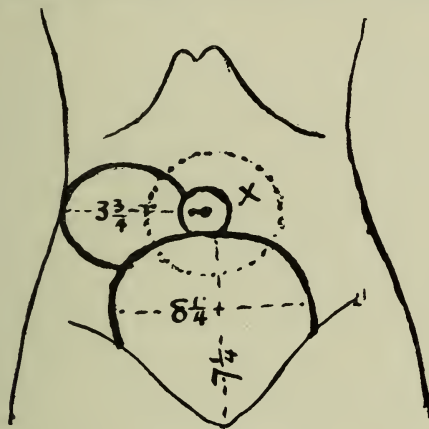
Mrs. R., aet. 41, nullipara. Admitted 17th April, 1899.

Report on Admission.—Patient last July took a very severe pain in the hypogastric and both iliac regions during the act of micturition. This pain was so severe that she had to keep her bed for some days afterwards and have hot fomentations and poultices applied. After that she was for a time in Ward II.

After the onset of the pain she observed a small swelling on the right side. It increased in size during her residence in Ward II., and has been gradually getting larger ever since, and spreading over the abdomen. There has been occasional retention of urine with very severe pain on micturition. Menstruation has been quite regular, but since the onset of above symptoms the discharge has been more profuse, and accompanied by pain in the lumbar region. Leucorrhoea has also been present. With the exception of palpitation of the heart the patient has always been healthy. Before admission patient suffered from retention of urine for three days, the catheter having to be passed. Last menstruation occurred last week, but just before admission, and at present patient

has a slight discharge. At the onset of the symptoms she had swollen feet and legs. There is no history of menorrhagia. Usually the period lasts three days and recurs at monthly intervals.

Physical Examination.—Patient is of a sallow complexion, thin, with furred tongue. Urine 1018, pale, and showing trace of albumen, with a few tube casts. Second cardiac sound is accentuated. Abdomen protuberant all over, but especially in the hypogastric region and in the right renal region. Palpation reveals a firm mass with irregular contour, which on deep palpation can be distinctly recognized as consisting of two



X Deep ovarian cyst.

parts corresponding to the bulgings of the abdomen evident on inspection. Percussion over hypogastric tumour is dull, but over portion in right side is clear, except in the centre, where it is perfectly dull. Tumour mass is movable, especially the portion in the right side, which can be displaced fully two inches towards the left. Just at the umbilicus is a very firm nodule projecting upwards between the mass in the hypogastrium and that in the right side. P.V. finger introduced into vagina comes at once upon a rounded mass filling the upper part of the vagina, the wall of which is continuous all round with the wall of the vagina. On the apex of this mass is a small circular opening resembling the os externum, but not surrounded by any portio vaginalis. Sound introduced through

this opening passes easily 3 inches in ordinary direction, leaving main mass of tumour behind it. Movement can be communicated to the whole mass. When the upper end of the tumour is drawn in one direction, the lower end passes in the other.

On May 3rd abdominal section was performed. Besides the myomatous tumours discovered by physical examination, the left ovary was found transformed into a cyst (tubo-ovarian) about the size of a large fist and somewhat irregular in contour, close above, behind, and to the left of the apex of the central tumour. Recovery was complicated with an attack of pneumonia, but patient left hospital for the Convalescent Home on 23rd May.

The relations and distribution of the tumour in this case were peculiar. The large mass which descended into the pelvis and had the cervix stretched along its anterior surface was apparently cervical in origin. The mass in the right lumbar region was chiefly the body of the uterus. The ovarian cyst lying mainly to the left of the vertebral column and below the level of the myomata was too deep in the abdomen to be detected on examination. The sound which passed 3 inches evidently only passed along a part of the cervix, and did not reach the body of the uterus at all. We are usually in the habit of exploring with a flexible bougie in these cases, but in this particular case we had not done so, and had therefore inferred that the right-sided tumour was a subserous myoma connected somewhat loosely with the uterus below, which we supposed the sound had entered.

CASE 9.

Myoma of uterus complicated with absence of one kidney and large calculi completely filling the other. Abdominal hysteromyomectomy. Death from suppression of urine.

M. M., aet. 27, unmarried. Admitted 11th May, 1898. Complaints of swelling of abdomen; first noticed about eleven

months ago. It was felt at first in the left side, and low down, but varies in position, being higher up in the mornings before she rises from bed. She does not think it is larger since it was first observed, but it is harder. Since this illness began she has got much thinner, and has lost colour. Except on micturition she has no pain, and it is at her periods that the pain is marked. During the last eleven months menstruation has come every two or three weeks, and has been excessive in amount, but is seldom painful, except when clots are passed. There is no offensive odour.

Physical Examination.—Urine alkaline, muddy, sp. gr. 1010, distinct albumen, considerable pus. Soft V.S. murmur at cardiac apex. Lower abdomen occupied by tumour, upper border of which is on a level with the umbilicus, firm but somewhat cystic in consistence—very freely movable both in vertical and horizontal directions, movement giving rise to some pain in left iliac region, where there is also some tenderness to pressure; vertically tumour can be raised into left hypochondrium. P.V. uterus and tumour are closely amalgamated, filling right side of pelvis and passing towards Douglas' pouch. Left fornix is free, and uterine cavity lies to left of and below the tumour.

At operation on 16th May the tumour was found to be an interstitial myoma of the anterior uterine wall. Uterus and appendages were removed, and stump stitched over and covered with peritoneum. For the first twenty-four hours 31 oz. of urine were passed, and except for a markedly feeble and rapid pulse, the patient seemed exceedingly favourable. In the next twenty-four hours only 16 oz. of urine were passed, but bowels were freely moved; and except that the pulse had become more rapid and feeble, there was nothing to suggest the perilous position of the patient. In the next twenty-four hours no urine whatever was passed, and the bladder was found empty when the catheter was used, and during the remaining thirty-four hours of life only 3 oz. were secreted. Hot poultices over the loins, tinct. digitalis, hypoderms of ether, strychnia, etc., oxygen inhalation at hourly and half-hourly intervals were all tried without effect. Except for

slight restlessness and frequent vomiting there were no nervous symptoms, and the patient gradually sank, the pulse becoming imperceptible for some time before death occurred.

We attributed the fatal issue to uraemia from suppression of urine, although there had been no distinctly uraemic symptoms. But the autopsy revealed a most remarkable condition of the urinary organs. "The right kidney was greatly enlarged, enormously distended in its pelvis and calices, and filled with numerous calculi, one of them being very large, and branched or lobulated. The left kidney was greatly atrophied (no secreting structure whatever was left, and the organ was hardly existent at all, J. K. K.), pelvis much distended, and the ureter at its junction with the pelvis is blocked by a group of small rough calculi and a villous tumour in which the calculi appear to be entangled. Bladder and stump of uterus present very healthy characters; the stump appears in process of healing."

The patient had evidently for some years been living with only one active kidney, and even this active kidney was completely occupied by calculous formation, over which the renal tissue was spread out in a thin layer. The calculus in this kidney might properly be called enormous, and we could not explain to ourselves how we had missed it in our examination of the abdomen. The pyuria we had attributed to cystitis instead of referring it to a renal condition, and had therefore probably examined the renal region less carefully than we ought.

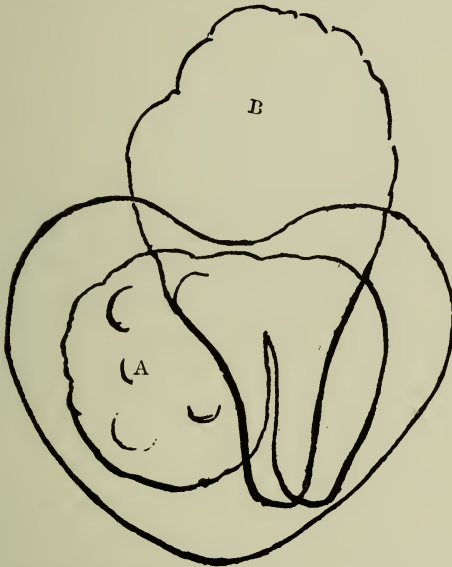
CASE 10.

Myoma of fundus uteri, assuming unusual position in pelvis. Abdominal myomectomy. Favourable course.

Mrs. J., aet. 29, nullipara. Admitted 19th October, 1898.

Report on Admission.—Patient is well nourished. Face rather dark in complexion, cheeks well flushed with blood. For the last year she has been in poor health, and at her menstrual periods has suffered from cramp in the abdomen.

Occasionally she has pain in the right side. The duration and amount of menstrual discharge have been diminishing of late. There is no leucorrhoea or intermenstrual discharge. For about the same time she has noticed "a lump in her womb," which has grown rapidly in the last six months. Patient thinks it has doubled in size in this time, but her doctor does not think there is much change since he saw it several months ago. Patient states that when she changes her position the tumour also changes, always falling over to the lower side.



A. Ordinary position.

B. Position when straightened.

Physical Examination.—In lower part of the abdomen, and chiefly to right of middle line, is a very hard mass with irregular surface reaching to about an inch and a half above the level of the umbilicus, while its lower border corresponds to Poupart's ligament. It measures longitudinally $5\frac{1}{4}$ inches, and transversely $4\frac{1}{2}$; its left border is an inch to left of middle line. It can be displaced laterally and without causing pain until its left border reaches $3\frac{1}{2}$ inches to left of middle line. It can also be displaced upwards till its upper border reaches 2 inches above level of umbilicus. This displacement is

effected without pain. P.V., the lower zone of the mass, which is irregular on the surface, is felt to occupy the right side of the pelvis, and is closely applied to the side of the uterus, the portio vaginalis being displaced towards the left pelvic wall. The sound passes $2\frac{1}{2}$ inches along the left side of the tumour.

On 24th October the tumour was removed. It was found to be connected with the fundus, and its apparent situation on the right side of the uterus was due to its bending completely over and lying along the right border of the uterus instead of, as usual, projecting upwards from the fundus into the abdomen. When it was raised out of the pelvis, the tumour and uterus were continuous in the vertical direction. This flexible character was perhaps connected with the peculiar structure of the myoma, which was not like any other I have seen. It was composed of softer and firmer parts, and the softer parts looked as if squeezed out here and there into masses projecting between the firmer strands, of which the bulk of the tumour was composed. On microscopic examination, however, the usual appearance of myoma was found.

CERTAIN FORMS OF OTITIS MEDIA ACUTA IN WHICH IT IS IMPORTANT TO MAKE EARLY PARACENTESIS OF THE TYMPANIC MEMBRANE.

By ROBERT FULLERTON, M.D.,

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ACUTE inflammations of the middle ear deserve our close consideration, not only from their frequency, but also from the dangerous results which so often follow.

For long the subject lingered neglected, but fortunately in these later years a deeper interest is being taken in the affection, and with a more accurate appreciation of the process the antiquated ideas which were formerly held are gradually being replaced by views more in accordance with the advanced position already attained in other departments of surgery. As already hinted, the consequences of acute otitis media may be so serious that anything that can be done to avert them ought to be taken advantage of, and I hope to show in this connection, that paracentesis of the tympanic membrane may be adopted as a prophylactic mode of procedure with a fair chance of success, as illustrated in the cases that follow. It should be understood, however, that I am dealing here only with cases of a so-called simple inflammatory nature, and not such as are met with complicating diphtheria, scarlet fever, etc., where the rapidly destructive character of the process is well known, and where paracentesis may be the means of preventing extensive loss of drum tissue.

To enable us to determine the cases in which paracentesis is specially called for, a general glance at the question is

necessary. It is a point open to argument whether in all cases of acute inflammation of the middle ear which has gone on to, or is likely to end in suppuration, paracentesis of the tympanic membrane should be resorted to, or whether in some the pus ought to be allowed to escape spontaneously through the drum.

In considering the question it should be borne in mind that all cases are not identical, and that even if similar at the onset they may be so modified in their course by the anatomical structures of the tympanic space that at least two distinct forms can be recognized. The form most generally met with is that in which the inflammatory process appears to affect the tympanic cavity as a whole, where, on looking into the internal meatus, the membrane, and it may be the tissues around its attachment, are seen to be more or less acutely congested. All characteristic points on the drum are lost, with perhaps the exception of the outline of the short process of the malleus, and outward bulging is usually observed in its lower portion. At an early stage in such cases hopes may be entertained of aborting the process by prompt employment of the means at our disposal to that end.

Should, however, our efforts in this direction be unsuccessful, the question of temporizing or of puncturing the drum is forced upon us. Before deciding on puncture it is well to pause and recollect that with spontaneous rupture relief from pain in many cases is more immediate than when an incision has been made, and that the after course, both as regards rapidity of resolution and power of hearing, is fully as satisfactory. Our decisions, I hold, must be governed by the appearance of the tympanic membrane. In cases where it presents a thinned and bulging look, and where the pain is either not excessive or can be controlled, it is better left alone, as rupture will soon take place. On the other hand, should it have a tough and thickened look—and in such instances we have usually marked constitutional disturbance with extreme pain—rupture is likely to be delayed, and steps ought to be taken to aid evacuation of the secretion. In regard to this, I am satisfied that if a paracentesis is to be made a general anaesthetic ought

to be administered unless otherwise contra-indicated. All our local anaesthetics act but imperfectly upon that highly sensitive and now inflamed area. Without general anaesthesia we submit our patients to an undesirable amount of excitement and suffering, and have further no guarantee of being permitted to perform with sufficient accuracy what is essentially a delicate piece of manipulation. It is true that without it we can incise the membrane, but from the invariable start given by the patient on the knife touching the surface of the drum we never know how much injury may be done.

In the form just considered the inflammatory process is most marked in, if not limited to the lower portion of the tympanum. With a Eustachian tube practically impervious, as is so often the case under the circumstances, and a resisting tympanic membrane, extension upwards and backwards might naturally be expected. As a matter of fact, however, spontaneous rupture of the drum may be counted on to take place before permanent mischief to the structures or extension backwards to the mastoid region occurs. When unfavourable results follow, they arise more from want of proper after-treatment than from what was left undone in the acute stage. Therefore, in performing paracentesis our object is practically limited to shortening, and that perhaps by very little, the pain of the sufferer.

When, as occasionally happens, the intensity of the process is confined to the upper portion of the tympanic cavity or that space whose lower limit may be regarded as about the level of the tensor tympani, the course is widely different, and it is to such I would direct attention.

To show how this portion may become a distinct space it is necessary only to refer briefly to the anatomical arrangements met with. Below the level of the short process of the malleus the tympanic space may be looked upon as vacant. From about that level backwards to the aditus ad antrum this space is encroached upon by the ossicles, their processes and ligaments, and the passage of the chorda tympani, in such a manner as to form a partition which under normal conditions permits of but limited communication. It would be out of

place here to enter minutely into the various connections existing between the tympanic membrane and ossicles on the one hand, and the tympanic walls on the other. These points, as investigated by Henle, von Tröltsch, and Helmholtz, are fully discussed by Schwalbe (*Anatomie des Ohres*). These structures tend to pass in a somewhat horizontal direction towards the short processes of the malleus and incus, thereby forming a more or less complete floor for the upper portion. But in addition to the generally recognized bands, adventitious striae are described, and Blake of Boston (Burnett's *System of Diseases of Throat, Nose, and Ear*, 1893) holds that such horizontal reduplications of the mucous lining are present in about 80 per cent. of human temporal bones. The striae are probably the remains of that gelatinous fibrous tissue which fills the tympanum and Eustachian tube at birth. I examined four healthy decalcified temporal bones in the Pathological Laboratory of the Royal Infirmary by making first a horizontal section below the level of the long process of the incus, and afterwards by successive thin horizontal slices through the tegmen tympani downwards. In three of these I found comparatively free communication between the attic and the lower tympanic space by means of an opening internal to and another external to the incus. In the fourth, the external mucous fold of the incus was prolonged forwards close to the posterior and superior ligaments of the malleus, shutting off the attic externally, whilst the internal mucous fold of the incus encroached so far on the space in that region as to leave but an extremely small elliptical slit, which led obliquely downwards and inwards. In this instance congestion of the lining membrane of the attic would in all probability have occluded the very limited communication which existed between the upper and lower portions of the tympanum. In all the four specimens which were examined the passage backwards into the mastoid antrum was unobstructed. When we bear in mind that all these structures are either clothed by, or are prolongations of the mucous lining, we can easily conceive how, when inflamed and swollen, they may act as an obstruction, *e.g.* fissures which formerly existed may become

obliterated by the tumidity, indeed there may probably be overlapping of the congested bands, and a study of the arrangement of the parts would lead one to expect that fluid pressing from above may be retained by a valve-like action on the part of these bands.

In the cases observed by me the ear affection was associated with a naso-pharyngeal catarrh, and the vaso-motor disturbance in the first instance probably affected the whole tympanic cavity. The normal balance of vital energy being thus impaired a disturbed working of the living tissues resulted and a condition was established favourable to the activity of micro-organisms whether present in the tympanic cavity or entering by the Eustachian tube. At the beginning the whole of the middle ear probably participated in the inflammatory action, but later the process appeared to concentrate its action on the upper portion, and the membrana flaccida became bulged outwards whilst marked hyperaemia was seen spreading up towards the segment of Rivini. At first the reaction is of a catarrhal nature, but purulency soon sets in and tension increases, as evinced by the bulging and by shooting pains. The inflammatory process spreads backwards to the neighbourhood of parts the implication of which we dread, such as the brain, lateral sinus, mastoid and facial nerve. The mastoid antrum is the part most frequently implicated, and I am inclined to think that, when such a result follows an acute suppuration of the middle ear, in a very large proportion of cases it arises from that form which we have just outlined. Unfortunately, in most instances of mastoid abscess following acute otitis the patient seeks advice only when the mischief has reached that stage when minute differentiation is impossible, and where there is well marked post-auricular swelling with or without discharge from the meatus. But the history given usually indicates that should there be a discharge from the meatus it has only appeared after a prolonged period of suffering, and perhaps only after the swelling over the mastoid had become marked.

But there is a sequel to acute suppuration of the middle ear and especially to the form we are considering, which, if not so

immediately alarming or dangerous as mastoid mischief, may ultimately prove both troublesome and serious in the highest degree. I allude to chronic suppuration with caries. We know that the lining membrane of the tympanic cavity acts also as a periosteum for the underlying bone, so that a catarrh of the middle ear is really a periostitis, and provided the inflammation be sufficiently intense or prolonged the tendency is towards destruction of the soft covering followed by superficial caries of the osseous structures beneath. According to Stacke (*Die Operative Freilegung der Mittelohrräume nach Ablösung der Ohrmuschel*, 1897), apart from affections of the ossicles, these carious patches are found most frequently on the roof of the tympanum or antrum. From what has been said we have thus an indication of the origin of troublesome cases of chronic suppuration associated with caries, always bearing in mind the important rôle which tubercle may play. Such cases exist without tubercle, and of these I shall single out examples of two types which are often encountered.

(1) When the greater part of the membrana tensa is destroyed, leaving the membrana flaccida intact. The ossicles are often present, and the handle of the malleus may be seen hanging free whilst posteriorly secretion more or less purulent in character is observed escaping from above. This may be regarded as the form resulting from a severe or neglected scarlatinal otitis, where the inflammation is very intense. I take it that here the process has acutely affected the whole tympanic cavity simultaneously, and by implication of the nutrient membrane of the ossicles or tympanic walls, caries has resulted. This same intensity of the process would in all probability soften and break down any barrier which may have existed between the upper and lower divisions, thereby affording free drainage for the discharge from above.

(2) Where there is suppuration in the attic with perforation of the membrana flaccida. This form is almost constantly associated with caries, the deafness is marked, and the secretion frequently so scanty that the lower part of the tympanic membrane looks dry and normal. Moreover, in most cases inflation of the middle ear by the Eustachian tube

produces no perforation whistle, thereby showing that communication does not exist. Here one would infer that the inflammation was essentially located in the attic, but for reasons associated with its character, and probably also from the anatomical arrangements present, the process had been such as to give rise to caries, only here the membrana flaccida had become perforated before extension backwards had taken place. For the nonce the posterior region has escaped, but it is exactly in such cases, when chronic, that we have most to dread its implication.

Having outlined this attic suppuration, from which I hold we have most to fear as regards both the immediate and the after consequences, I shall now give three cases which are fairly typical.

CASE 1.

M. K., aged 8. This lad was seen in consultation on 11th October, 1897. About a week before he had caught a bad cold, and for the last two or three days had complained of pain in his right ear. The night before I saw him this pain was very severe and shooting in character. He looked pale and tired. His pulse was 100. The temperature was not taken. There was great tenderness over the mastoid antrum and mastoid region generally, but no swelling could be seen. His pharynx looked red and inflamed. The right tympanum showed a slight degree of hyperaemia. The left tympanic membrane was found to be bulging in its upper portion, especially anteriorly, where it was also very red. The lower portion looked grey and indrawn. On this side the watch seemed to be heard on contact. On the 12th October, as the symptoms had not abated, the drum was incised at its upper and anterior part, through the bulge, as far as to the periphery. Syringing brought away a good deal of blood with shreds of mucus. In the evening I found a profuse discharge coming from the ear, but the patient was feeling better, his pulse being now 90 and his temperature 99°. The pain over the antrum was less, but still very marked lower down over the

mastoid. The bulging of the membrane had subsided and the short process of the malleus could be distinguished. On seeing the patient again on the 3rd November I learned that the ear had discharged profusely for a week after being punctured. The membrane had now healed and the redness was much less, but still tenderness on pressure was felt over the lower part of the mastoid portion. I did not feel confident as to the result and asked that he should be brought to me again in a few days. When I saw him five days later I found that the membrane had cleared up considerably since last visit, and no pain could be elicited by pressure behind the auricle. The patient called again on the 21st December, when the tympanic membrane was found to be movable and free from redness, and the watch could be heard at a distance of six feet, which is little short of normal. I learned in spring 1899 that this satisfactory state had been maintained.

CASE 2.

H. L., aged 13. Was seen on 7th January, 1899. The history given by the family physician was that the patient had returned from school fourteen days before, feeling unwell. His temperature was 103° when seen by him at that time, but had gradually come down to normal, and the patient was apparently quite well until four days before being seen by me, when he developed a violent cold in the head through standing at an open window. I found the patient very restless and irritable, and with a temperature of 102° . For the last two days he had complained of pain in his right ear, which was much worse at night, coming on in paroxysms, shooting up from the ear over the back of the head. There was marked tenderness on pressing over the antrum and mastoid process. The upper portion of the tympanic membrane was found to be red and bulging, especially posteriorly. With the exception of some hyperaemia along the handle of the malleus, the lower portion seemed unaffected. The watch was heard on contact. The left ear appeared normal.

A spray was ordered for the naso-pharynx and leeches applied behind the auricle. I saw the patient again on the 11th, when pain was felt on pressing behind the auricle, and the temperature was 100°. The upper portion of the drum was bulging and had a sodden reddish look. The lower was deeply indrawn. After carefully cleansing the drum a mixture of cocaine, carbolic acid, and menthol was applied on cotton wool and an incision afterwards made from the short process posteriorly to the periphery. This was followed by a bloody serous discharge. The local anaesthetic seemed to have little effect. I did not see the patient again until 19th January, when I learned that for the first four days after puncturing a profuse muco-purulent discharge had come away from the ear with relief to the pain. The discharge had then ceased for two days, when it returned again, since which time the amount had been irregular. The discharge was small in amount. The membrane was red and bulging as when first seen, the wound having healed over except a pin-point opening anteriorly, through which a few drops of fluid escaped on suction. The temperature was 99.4° and the pain behind the auricle less than formerly, but great tenderness to touch was complained of above its upper attachment. On the following day the temperature was 100°, and the patient had had severe pain during the night. There was marked tenderness all round the attachment of the auricle and slight oozing from the pin-point opening in the drum. The patient being under chloroform an incision was made along the line of the former cut. When seen on the 30th January, after Politzerization on several occasions, the drum was found to be healed, tenderness on pressure had disappeared, the Eustachian tube was free, and the watch could be heard five feet from the ear. He made steady progress, and when seen for the last time on the 2nd May—after having been back at school for two months—the watch could be heard five feet from the ear, and the drum looked normal except for the trace of a cicatrix.

CASE 3.

This case was that of J. H., a gentleman about 50 years of age, who had been a long time in the East Indies and was in the habit of taking large quantities of quinine³ for malarial fever. He was seen on the 30th January, 1899, when recovering from an attack of influenza in which his temperature had been as high as 106°. For the last few days he had complained of severe pain in the right ear, and the night before I saw him the pain was so acute that injections of morphia had to be given. It was shooting in character and spread from the ear over the head. The patient was very anaemic, and his temperature was 100·5°. The right tympanic membrane was seen bulging in its upper and posterior portion, but was not very red. The watch could not be heard on pressing it closely against the ear, and there was marked tenderness over the mastoid region. I saw the patient on the following day (31st January), and, finding no improvement in his condition, slit the membrane horizontally backwards from behind the short process to the periphery. For this cocaine was used, but the pain was intense. A free discharge of bloody serous fluid came away. I saw the patient again on the 8th February, when I was told there had been a copious discharge for five days after it had been incised. The membrane was now healed and no pain felt on pressing over the mastoid region, but in front of the tragus there was marked tenderness, which I attributed to the presence of a small furuncular swelling in the meatus. He still could not hear the watch on contact. I did not see the patient after this, but subsequently learned from his family physician that, after making a good recovery and being out and about, he contracted a chill and died of pneumonia.

In the cases just given the inflammatory focus lay in the attic portion of the tympanum, and there were indications of acute congestion of the mastoid region. I think it may be claimed that, by performing paracentesis here at

a comparatively early stage, the hearing power was preserved, mastoid suppuration aborted, or possibly the patient spared a prolonged chronic suppuration. The converse of this sequence of events was supplied by the case of a man, 48 years of age, where unfortunately no notes are available, but in which bulging of the upper and posterior part of the tympanic membrane of one side, with marked deafness, was observed for weeks. He had practically no pain, and but faint signs of inflammation were noted on examining the ear. He is now permanently deaf on that side, the process having insidiously spread backwards, causing a mastoid abscess and necessitating an operation.

ON THE TREATMENT OF CICATRICIAL CONTRACTION OF THE FINGERS BY TRANSPLANTATION OF COMPLETELY SEPARATED SKIN FLAPS.

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THE ultimate effect of the destruction of a sufficiently extensive area of the cutis vera on the flexor aspect of a joint is well known. The replacement of the normal tissue by a cicatrix gives rise, as the latter contracts, to a permanent flexion. In no part of the body is this result better marked than in the case of the fingers. Destruction of the entire thickness of the skin on the flexor surface of a finger, brought about by say a burn of the fourth or higher degree, if left to heal by the natural process, is certainly followed by a very marked flexion of the finger. This contraction, if the damage involves the greater part of the flexor surface of the finger, and perhaps a small part of the surface of the palm, will become so extreme that the finger will be brought into contact with the palm, and fixed there by dense cicatricial tissue.

A condition such as this is one which involves much interference with the normal use of the hand, and becomes a very serious matter in the case of those who require or will require to earn their livelihood by manual labour. The removal of a deformity of this kind is, therefore, a matter of very considerable importance to the patient.

Fixation of the fingers in the extended position while healing is in progress, if indeed always possible, can scarcely

be said to be effective in preventing the formation of the deformity, as the contraction which ultimately takes place in the scar, after removal of the splint, will flex the finger as much as if the precaution had not been taken. The object of this paper is, however, to consider what line of treatment should be adopted in cases in which the cicatricial contraction is already present, and in which the wound is entirely healed.

The method of treatment by simple section of the cicatrix and forcible extension of the finger will be followed by recurrence of the contraction, and this recurrence will not be prevented as an ultimate result, although the finger is kept extended, until the wound is covered with epithelium, and although the process is hastened by small grafts of epithelium. The only result which can be expected from this treatment is that on liberation of the finger from the extending force, it will gradually return to its condition of extreme flexion.

To prevent the recurrence of the condition, an operation of a more radical nature is required, one which will replace the cicatricial area by a tissue which will not contract. The plastic operation of separating up the cicatrix, and mobilizing flaps of sound skin in the neighbourhood to the extent required to allow of extension of the part, might be applied in the case of the fingers. In this case the flap of sound skin would require to be taken from the palm, as in Busch's operation for contraction of the palmar fascia; but the amount of separation which would be required before the finger could be extended, would be so great that it would simply be a case of grafting with the graft taken from the palm.

The method which has been employed in the cases here recorded, is that of transplantation of tissue from a distant part to replace the cicatrix, previously completely dissected off. The older method of grafting of small portions of epithelium was not tried, as this does not prevent subsequent contraction. A single strip of epithelium sufficiently large to cover entirely the raw surface left after removal of the

cicatrix (Thiersch's grafting) was tried, but, as the report will show, with unfavourable result, although the graft healed in perfectly. The method which did give good results was that of transplantation of the entire thickness of the skin, a method employed by Wolfe of Glasgow in 1875 for the removal of ectropion, and recently advocated by Krause of Altona, who, by improving the technique, has done much to ensure the success of this operation.

CASE 1.

Contraction of little finger, due to cicatrix of burn. Excision of cicatrix and transplantation of skin. Failure.

A. M., female, aged $1\frac{1}{2}$ years, was brought to the Victoria Infirmary Dispensary on 28th November, 1893. The little finger of the right hand was bound down to the palm by a dense cicatrix, the result of a burn received some months previously. Under chloroform the cicatrix was divided transversely at several points, and the finger forcibly extended. This was accomplished without dividing the tendons, but the sheath of the tendons was in great part exposed, the divided cicatricial tissue not being more than sufficient to form one or two bands stretching across the sheath. The entire cicatricial area was then excised, leaving a wound surrounded by healthy skin, and having for its floor nothing but the sheath of the tendons, and extending throughout the entire flexor surface of the finger, and projecting also for a short distance into the palm. A lenticular area of skin of sufficient size to cover the wound completely was excised from the forearm, being dissected off free from adipose tissue, and transferred to the wound on the finger, where it was fixed in place by a few sutures of horse hair. The finger was then covered with a piece of perforated green protective silk and dressed. After the wound in the forearm had been sutured, a forearm splint was applied. This operation was performed in the out-patient department, and the child was brought back twice weekly for inspection.

At the end of ten days the dressings were removed to ascertain the result, and it was found that the transplanted piece of tissue was living and looking red and healthy, although the superficial corneous layer of the skin was soft and separating. Dressings and splint were reapplied. When the child was next seen at the end of the fourteenth day, it was found that the dressings had been removed and reapplied by the mother, in order to show the interesting result to the child's father. On inspection of the result, the grafted piece of skin was found separated and lying in a collection of pus, while the surface of the finger was covered with granulations. The child was seen some months later, when the original condition of contraction had been completely reproduced.

CASE 2.

*Contraction of index, middle, and ring fingers due to cicatrix of burn.
Excision of cicatrix and transplantation of skin. Success.*

D. M., male, aged 2 years, was admitted to Dr. Patterson's wards in the Western Infirmary on 2nd November, 1897. When patient was six months old he sustained a burn on the flexor surfaces of the second, third, and fourth digits, and over part of the palm of the right hand. The burn was caused by the child grasping a red hot cinder. When the burn was healing the three fingers were being gradually drawn towards the palm, and by the time that cicatrization was complete, the fingers had been drawn almost completely into the palm. When the patient was first seen, a year and a half after the accident, the following was the condition found :

The right hand presented the index, middle, and ring fingers flexed to the extent of being in close contact with the palm. They were held in this position by tense indurated bands of cicatricial tissue, which were so rigid as to prevent any appreciable extension, even on considerable force being used. The cicatricial bands, fused in the palm, extended from the lower third of the palm to the terminal phalanges of the

fingers. The fingers also presented a degree of webbing throughout the extent of their first internodes.

On 5th November, 1897, under chloroform, and after rendering the limb bloodless, the cicatrices were divided transversely at several points, and the three fingers forcibly extended. This was effected without division of the flexor tendons, and the incisions in the cicatrices were torn open to the depth of the tendon sheaths. Next, the entire cicatricial area on the three fingers and palm was dissected off, the result being that on the flexor surface of each finger there was a wound left, bordered by healthy skin and having for floor the sheath of the tendons, while the wound in the palm had for floor adipose tissue. Next, the tourniquet was removed, and bleeding arrested by torsion, and the capillary bleeding by pressure. While this was being done, the skin flaps were cut from the forearm. Two lenticular flaps of sufficient size, each to cover a finger and portion of the palm, were cut, taking the entire thickness of the skin, and dissecting the flap from the underlying adipose tissue. These two flaps were transferred to the index and ring fingers, being laid on the tendon sheaths, and fixed by several points of horse hair suture to the healthy skin around. The entire extent of the wounds on these two fingers and corresponding parts of the palm was thus covered over. In order to cover the middle finger, and for the sake of comparison of the value of the two methods of grafting, a Thiersch's graft was cut of the requisite size from the forearm, and laid over the wound on the finger and corresponding part of the palm. When the operation was completed, the entire wound on the three fingers and palm was covered over. The grafts were then covered with perforated green protective silk and dressings applied. The two areas on the forearm from which the whole skin grafts had been cut, were now closed completely by silk-worm gut sutures. The edges of the skin came easily together, after the skin on either side had been dissected up for a short distance. The limb was then fixed on a forearm splint.

The first change of dressings was made at the end of fourteen days. All the grafts were found to have taken. Minute

portions at the two extreme ends of the skin grafts were dead and separating, but with this exception the grafts were in a healthy condition, presenting a red colour and adhering firmly to the fingers. The redness of the surface was due to the separation of the superficial layers of the epidermis, which lay in the dressings as a soft debris.

The dressings and splint were applied for another fortnight, and, when removed at the end of that time, the grafts were found firmly healed in, the surface of the whole skin grafts presenting a normal skin surface of a redder tint, however, than is natural.

As a precaution the hand was covered up for another fortnight, at the end of which time the child was allowed to use the hand.

The patient was seen on 6th January, 1898, two months after the operation, and it was then found that while the index and ring fingers remained perfectly straight, the middle finger, which had been covered with the Thiersch's graft, was tending to contraction. The graft on the index finger was perfectly outlined by a cicatricial line, and presented the appearances of normal skin, and had acquired the transverse lines at the joints.

This patient was again admitted to hospital on 28th June, 1898. The condition of the hand then, about seven months after the operation, was as follows (Fig. 1): The contraction of the middle finger (Thiersch's graft) had recurred completely, the finger being bound down firmly into the palm. The index finger remained straight, while the ring finger was semiflexed, being drawn down chiefly by adhesions to the middle finger.

On 7th July, 1898, the cicatricial tissue was removed from the flexor surface of the middle finger, and also the portion of cicatrix which involved the ring finger. Whole skin grafts were cut from the thigh and stitched into the gaps on the fingers and palm. The fingers were then dressed with gauze, green silk protective being avoided. The limb was then fixed on a splint.

The dressings were not disturbed till the end of five weeks, when it was found on inspection that both grafts had taken

perfectly. The skin had a normal appearance, and the fingers were perfectly straight, and the patient was allowed to use the hand.

This patient was again examined on 26th August, 1899, *i.e.*, about twenty-two months after the first, and fourteen months after the second operation. The result had proved entirely successful (Figs. 2 and 3). All three fingers could be voluntarily extended to almost their normal degree, and flexed also fully into the palm. The use of the hand was thus entirely regained, and very little contraction had occurred in the transplanted flaps. The flaps had a somewhat scaly, and at the margins a somewhat cicatricial aspect, but otherwise appeared to fulfil the requirements of the flexor surface of the fingers. Sensation over the entire transplanted part was apparently normal, as tested by pricking with a needle. On the surface of the grafts minute hairs were visible by the aid of a lens. The grasp of the hand was as good as that of the other hand, and the child made constant use of the hand in a perfectly normal manner.

CASE 3.

Contraction of ring and little fingers from cicatrix of burn. Excision of cicatrix and transplantation of skin. Success.

D. C., male, aged 12 years, was admitted to Dr. Patterson's wards in the Western Infirmary on 3rd June, 1898.

When patient was one year old he met with an accident to his right arm, for which the arm was fixed in a sling, which was pinned round the hand to prevent movement. While the arm was thus fixed, he set fire to a box of matches which fell inside the sling, and inflicted a severe burn on the ring and little fingers. The burn was treated without any particular precautions to prevent contraction, and when the dressings were removed it was found that both fingers were fully flexed by cicatricial adhesions into the palm.

His condition on admission to hospital was the following: The little finger was fixed by dense cicatricial adhesions closely

to the palm. These adhesions extended from the palm to the tip of the finger, the entire length of the finger being practically fused with the palm. The finger was also markedly shortened, and had undergone a movement of rotation, due to the contraction of cicatricial tissue on the inside of the finger and palm, to the extent that the nail presented on the inner aspect. The ring finger was also firmly bound down to the palm by cicatricial adhesions, extending from the palm along the finger to its terminal phalanx.

On 9th June, 1898, the cicatrices were divided and attempts made to extend the fingers. This, however, could not be effected on account of contraction of the tendons. The tendons were therefore lengthened at one of the nodes where the sheath is deficient, and the tendons therefore easily accessible. This was done by a method which allows of lengthening without severance of continuity. When the fingers by this means had been brought to the fully extended position, an attempt was made to remedy the rotatory displacement of the little finger, but this was found to be impossible. The entire cicatricial area was then excised, and when this was completed the tendon sheaths in the fingers were quite denuded, the entire breadth of the flexor surface having been removed. Two grafts of the whole thickness of the skin, and of the requisite size completely to cover over the defect, were cut from the front of the thigh and implanted on the fingers, being fixed in place by horse hair sutures. The grafts were covered with perforated green silk protective and dressed, and the forearm and hand fixed on a splint.

At the end of seventeen days the dressings were removed for the first time, and it was found that both grafts had taken throughout their whole extent, with the exception of two minute portions at the ends of the ring-finger graft. The stitches were removed. The superficial corneous layer was soft and separating, but beneath that the graft showed a normal skin surface. The dressings were reapplied, omitting the green silk protective, and the patient was dismissed from hospital, to return as an out-patient.

Ten days later the part was dressed for the second time.

Both grafts were found healing well, and presented corrugated surfaces which were dry and of the colour of normal skin. There was a small raw surface at the proximal end of the ring-finger graft.

The case was again seen at the end of July, seven weeks after the operation, when the fingers were found straight and the grafts presenting the characters of normal skin, and looking so well that the boy was allowed to use the hand.

This patient was examined again in July, 1899, thirteen months after the operation. The condition then was as follows (Figs. 4 and 5): Contraction had not recurred at all in the ring finger, and very slightly, if at all, in the little finger. The ring finger could be voluntarily completely extended and flexed into contact with the palm, but it could not make firm pressure on the palm. The little finger could be voluntarily extended and flexed, but there still of course remained the rotatory displacement, which was not remedied at the operation, and it still remained somewhat shortened. The use of his hand had been gradually improving during the year, and he used both fingers in grasping, and could grasp a body $\frac{3}{4}$ inch in diameter firmly with them. The grafts showed the transverse markings at the flexures, which had been assumed by the grafted skin. The cicatricial lines round the margins of the grafts were evident. The surfaces of the grafts were of the same colour as the flexor surfaces of the other fingers, but here and there showed a somewhat scaly appearance. Sensation of touch and pain was normal throughout the extent of the grafted portions, and the grafts were movable, as normal flexor skin surfaces, on the tendon sheaths below, but grasped from side to side they felt somewhat denser.

The first point which may be considered in connection with the above cases is the comparative value of Thiersch's grafts of epidermis, and of transplantation of the entire thickness of the skin. The former method was employed only in the case of a single finger, and of course there is an objection to arguing from a single case. However, in that case the

conditions were entirely favourable as a test of the value of Thiersch's grafts; for the grafted epidermis healed in perfectly without reaction of any kind. Despite this favourable progress, the finger grafted by this method showed at the end of two months a distinct tendency to reproduction of the contraction, while the two neighbouring fingers remained perfectly free from contraction. This reproduction of the contraction continued progressively, until when seen at the end of seven months the contraction of the finger was as extreme as before the operation, while no contraction had yet occurred in the two neighbouring whole skin grafts, although the ring finger appeared semiflexed from adhesions to the middle finger (Fig. 1). On general considerations also this result of Thiersch's grafting is not surprising. The graft represents merely a covering of epithelium, and the important structures present in the cutis vera have nothing to represent them. The structure therefore of the new flexor surface cannot be other than that of cicatricial tissue, and, therefore, must be expected to undergo much contraction.

With regard to the transplantation of the entire skin, the cases show that if the immediate result of this operation is successful, the ultimate result is also good. The new flexor surface does not tend to undergo contraction to any appreciable degree, and approaches in characters as nearly as could be expected to the normal tissue. The grafts appear and feel like normal skin, and soon take on the normal markings at the flexures of the joints. Sensation also is developed in them, apparently as perfectly as if they were the natural covering of the part. With the exception of the first case, all the skin grafts healed in without trouble, although the surface on which they were implanted, viz., the naked tendon sheath, did not look like one best fitted for the nourishment of portions of tissue so thick.

In carrying out these operations, the use of antiseptics was limited to the thorough sterilization of the skin before operation. The application of antiseptics to the wounds ought to be avoided, as the vitality of the tissues may thus be lowered below that which is necessary for success of the grafts. The

entire cicatricial area is removed, and, if the tendons have undergone contraction from the long duration of the flexed position, these must also be divided or lengthened before anything further can be done. In the case in which this was necessary, eleven years had elapsed from the date of accident, while in the other two cases, in which respectively one year and eighteen months only had elapsed, the tendons did not resist extension after removal of the cicatrix.

Haemorrhage ought to be arrested before applying the grafts, and if the scar is dissected off while the hand is bloodless, the tourniquet ought to be removed immediately this is effected, in order to give time for the arrest of haemorrhage while the graft is being cut. In preparing these grafts, I have never removed the adipose tissue with them, but have always outlined the lenticular area to the depth of the adipose tissue, and then dissected the skin off. There has never been any difficulty in closing the wound left after removing the graft, although the adipose tissue was left, all that was found necessary being to mobilize the skin for a short distance in the vicinity. The graft should be of the requisite size exactly to fill the gap presenting on the finger, so as to leave no surface to heal by granulation, and also not to overlap the surrounding skin at any point. These grafts are usually simply laid on the part to be grafted, but in the case of the fingers, it will not be found easy to keep them in place without suturing. The smooth tendon sheath affords no hold for the graft, and the edges also of a strip of skin so narrow cannot well otherwise be prevented from rolling inwards. The application of a few horse hair sutures kept the grafts in perfect position, and did not prevent even the portion of the graft transfixed from retaining its vitality and healing.

The green silk protective, employed in all except the second operation in Case 2, was used to prevent the dressings from adhering to the grafts and displacing them when being removed. I think, however, that this method of dressing is unfavourable to the grafts. Although perforated, the impervious tissue kept the part always moist, a condition favourable for sepsis, and also doubtless accounting for the separation

of the corneous layer of the epidermis. This separation and breaking up of the corneous layer of the epidermis is a danger, as micro-organisms imprisoned in the superficial layers, which might have resisted the sterilization of the skin, are thereby rendered effective. In the case in which the protective silk was not used, and the part dressed simply with gauze, the corneous layer of the epidermis did not break up and separate. Of course, if a dry dressing is employed, it ought to be left on until it is certain that the grafts have firmly healed in, as any attempt to remove it will be almost sure to tear the delicate early adhesions of the graft. In the case in which it was tried, the grafts were so firmly fixed at the end of five weeks, when the first examination was made, that the part did not require to be dressed again. If elevation of the patient's temperature indicated anything wrong in the early stages of healing, it would of course be better that protective silk had been used, as the dressing would be easier to remove. But even in that case, the very fact of something having gone sufficiently wrong locally to raise the temperature would probably in itself be fatal to the vitality of the grafts.

The question of the ultimate fate of the grafts from a histological point of view has been lately the subject of several investigations. According to Enderlen¹ the transplanted tissue gradually is removed and replaced by growth from below, and by the end of four weeks the grafts are almost entirely replaced. From a clinical point of view, however, the advantages of using grafts of the entire skin, as compared with those of epidermis only, are well established. The difference amounts to the difference between a normal skin surface and a cicatrix. The description, therefore, which Enderlen has given of the histological process is not what we should expect to be the case in consideration of the retention by the grafted piece of skin of the characters of skin. Thus, grafts cut from the skin of the thigh retain their hair follicles, as shown by the growth of fine hairs in the graft, although the part grafted, as in the case of the palmar surface, is normally destitute of

¹ Enderlen, "Ueber das Verhalten der elastischen Fasern in Hautpflanzungen," Virchow's *Jahresbericht*, 1898, i., p. 54.

hair. In view of this Braun¹ has recently studied the microscopic appearances of several of Krause's cases of grafting by this method. Small portions of the grafted skin at various stages were excised for examination, and he found that the structures of the transplanted cutis were almost *in toto* retained. The elastic and fibrillar tissue, papillae, hair follicles, glands, and muscle cells were all found to be retained at periods ranging from four days to three and a quarter years from the date of the operation. These also could not have been replaced from below, as the graft was found demarcated from the underlying parts by a true cicatricial tissue. This view is supported by Henle,² who describes the process of healing from experimental grafting in rabbits as follows: The early nutrition of the transplanted tissue is maintained entirely by transudation, but by the fourth day, as shown by injected preparations, the vessels of the graft are already included in the circulation. This is effected by outgrowth of capillary vessels from those of the tissue below the graft, which make connections with those of the graft. After this stage the healing process is simply that of healing *per primam*. The view thus expressed by Braun and by Henle is in accordance with clinical experience, and explains the superiority of grafts including the cutis vera over those which consist merely of epidermis.

In conclusion, I have to express my thanks to Dr. Patterson for allowing me to perform the operations in Cases 2 and 3 in his wards in the Western Infirmary.

¹ Braun, "Histologische Untersuchungen über die Anheilung Krause'scher Hautlappen," Langenbeck's *Archiv*, 1899, vol. lix., p. 340.

² Henle, "Verhandlungen der deutschen Gesellschaft für Chirurgie: XXVIII. Kongress," *Beilage zum Centralblatt für Chirurgie*, 1899, No. 27, p. 30.

PLATE I.

FIG. 1.



FIG. 2.



FIG. 3.



FIG. 4.



FIG. 5.



EXPLANATION OF PLATES.

PLATE I.

FIG. 1. *Case 2.* Photograph of hand seven months after the first operation. The index finger still remains perfectly uncontracted, and the outlines of the transplanted skin flap are indicated. The ring finger is shown partially flexed, but this is caused chiefly by an adhesion to the middle finger. This finger also was grafted with a skin flap. The middle finger shows the condition of contraction completely reproduced, and this is the finger which was treated by Thiersch's epidermis graft.

FIGS. 2 and 3. *Case 2.* Hand, twenty-two months after the first, and fourteen months after the second operation. The index finger, which was not interfered with at the second operation, still remains uncontracted. The middle finger, which was treated with a transplanted skin flap, now remains almost uncontracted, and the same is the case with the ring finger. Fig. 2 shows the amount of voluntary extension, and Fig. 3 that of voluntary flexion.

PLATE II.

FIGS. 4 and 5. *Case 3.* Hand thirteen months after the operation. The grafted flaps on the ring and little fingers have in the photograph a somewhat cicatricial appearance, but this is due to the scaly quality of the skin. The little finger shows the shortening and rotatory displacement which were present before operation and which could not be remedied. Fig. 4 shows the degree of voluntary extension, and Fig. 5 that of voluntary flexion.

ON TESTING THE HEARING WITH HIGH AND LOW NOTES IN DISEASES OF THE EAR.

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IN this paper I propose to discuss briefly the testing of the hearing powers for high and low notes by air-conduction. In recent times the importance of this means of diagnosis has become more evident, and it now takes rank as one of the most valuable aids in the differential diagnosis between diseases of the sound-conducting and of the sound-perceiving apparatus.

Lucae was the first to draw attention to the value of testing the hearing with notes of low pitch, and it is to him, and more recently to Bezold, that the introduction of this means of diagnosis is due.

The latter has elaborated the means of examination, and is now able with his instrumentarium to produce any tone within the limits of hearing. The instruments he uses are, ten tuning-forks in ascending series, two covered organ-pipes, and Galton's whistle.

For testing the hearing powers in the lower parts of the scale, tuning-forks are essential, though expensive. No other instrument is so free from overtones, and even when these are present with tuning-forks they are far removed from the fundamental, and are therefore unlikely to cause confusion; and further, they die away so rapidly that by waiting for a few moments after the instrument has been bowed or struck, the fundamental will alone be heard.

Testing the hearing capacity for high notes is a more uncertain procedure than testing it for the low notes. There are two reasons for this: in the first place, the instruments employed necessitate the production of accompanying sounds in addition to the particular tone with which we wish to test the hearing. Thus, in using Galton's whistle it is, even for practised ears, by no means always easy to say when the high note becomes lost and replaced by the highly pitched hiss of the air rushing past. With König's cylinders there is not quite so much difficulty, because the metallic ring of the hammer against the steel is unmistakably lower than the note excited by the longitudinal vibration of the rod. But even with this instrument the patient must have a good idea of what he is to listen for. This is best done by sounding one of the lower pitched cylinders, the note of which he can undoubtedly hear and distinguish from the accompanying metallic ring of the steel, and then proceeding up the scale he will be able to say when the proper note of the cylinder is no longer heard.

In the second place, the pitch of the highest audible note varies greatly in individuals whose hearing may still be considered normal. We are, therefore, not justified in assuming the existence of any real pathological condition, unless the patient is unable to perceive notes as low as 15,000 to 12,000 vibrations per second. For this purpose I have constructed short brass bars which give approximately from 8,000 to 12,000 vibrations per second. They are suspended at each end by a cord, and are excited by drawing a well-resined violin bow across them. The note produced is very high and correspondingly disagreeable, but is much louder than that excited by either Galton's whistle or König's cylinders, and the insignificant noise of the rubbing of the bow which accompanies the note could not cause confusion. Of course the note is only produced as long as the bow is being drawn across the cylinder, for rods of such high pitch cannot continue their vibrations for any appreciable period after the exciting force has been withdrawn in the way that rods of lower pitch, such

as tuning-forks, can. The vibrations of the rods are transverse, and not longitudinal like those produced by König's cylinders.

In testing the hearing power for the lower notes it is not necessary to close either ear, but in the case of the higher notes the ear which is not being tested should be firmly closed or fallacies will occur.

As regards the value of the results obtained by testing the hearing for different parts of the scale, from my own observations I would unhesitatingly say that the examination by the low notes gives us the most reliable information, and when we come to consider the principle upon which the testing depends, the reason will be clear. Out of fifty cases of middle-ear disease, in which I tested with the low-toned tuning-forks, I found that in only one case was there no loss of perception for some of the low notes. This was the case of a man who worked in a room where copper-boilers were made, and there was a loud noise constantly going on. The results of treatment seemed to indicate that it was a case of middle-ear disease, in spite of the fact that he heard the notes down the lowest part of the audible scale.

On the other hand, although I have found that the hearing for high tones is lost first in most cases of deafness due to affections of the sound-perceiving apparatus, yet I have seen not a few exceptions to this rule. Moreover, when the deafness is due to disease in the central nervous system, or in the trunk of the auditory nerve, the rule does not hold at all. Thus, in a very interesting case observed by Christ and Siebenmann ("Ueber d. Central. Hörbahn," etc., *Zeitschr. f. Ohrenh.*, Bd. xxix., s. 81), the high notes were perceived up to 1.5 on Galton's whistle, that is, almost to the normal extent, whereas the notes below A_{-1} , an octave and a half, were lost.

There is no doubt, however, that in affections of the cochlea it is a very general rule that the upper notes are lost first, and several patients in giving the history of their complaint volunteered the remark that the first time they were

aware of any deafness was when they found they did not hear the singing of the lark while companions present at the time were able to do so. The most interesting of these cases is the following.

Two or three years before seeing me the patient suffered from a very severe attack of pneumonia following influenza. His physician despaired of his life, but recovery took place after eleven days' illness. The shock of the disease, however, he had not got over when I saw him, and it is doubtful if at his age (53) he ever will get quite over it. He has, moreover, suffered from considerable business worries since his illness.

When I first saw him I observed that his hair had turned white in patches, and he informed me that this first appeared about a year after the pneumonia. Two years after the pneumonia he became aware of the fact that he was deaf when walking in the country with his wife, who remarked that she heard the lark singing, he himself being unable to hear it. A year later than this he complained of aching in his eyes, and on examination by an oculist it was found that he was almost blind in the left eye, the condition being diagnosed as thrombosis of the vein. The right eye was healthy.

On examination of the ear I found that he was deaf for notes above a^5 (6826 *v.d.*), and he only heard faintly those for some considerable distance lower in the scale even when they were sounded loudly. (The note of the lark is of course lower than a^5 , but it must be remembered that when the bird is singing at some distance the sound is faint.) The membrane was normal and the bone conduction diminished.

Since that time the affection of the ears does not seem to have progressed. He can hear the thrush and the blackbird easily, and in conversation it would not be suspected that he is deaf. The hair has returned to its original colour, but the eye appears to be in the same condition as it was when he was first examined.

Occasionally when testing the hearing power we find gaps in the scale; that is, the patient may be unable to hear a

series of tones in more or less close proximity while he is still able to hear tones both higher and lower in pitch. Such a condition indicates an affection either of the cochlea or of the nerve-paths, but so far as I am aware is never found in diseases of the middle ear alone.

Cases of this description are of extreme interest if a post-mortem examination of the organ of hearing can be obtained, because it is from such, examined carefully during life, and then again post-mortem, that a knowledge of the functions of the labyrinth is established. Two cases of this description have recently been described very minutely by Bezold (*Ueber d. funkt. Prüf. d. menschl. Gehör*, s. 173), and they seem to support the view that sound is analysed in the cochlea, although perhaps the author goes too far in assuming that this analysis takes place in the way suggested by Helmholtz.

On account of the importance of the information to be derived from the microscopical examination of the organ of hearing in persons who have been examined during life and found deaf to certain portions of the musical scale, I have added a note at the end of this paper describing a method of preparing sections of the cochlea. After a considerable experience in different methods of making such preparations, I have found it the only one that can be relied upon to give satisfactory results in the *human* subject; there may be others, but I do not know of them.

The causes which bring about loss of hearing in different parts of the scale are, I believe, different according to the part of the scale for which the hearing is lost. Taking that group of cases in which the higher notes are lost, it has been suggested that in these there is destruction of the organ of Corti or the nerve-fibres in the lowest whorl of the cochlea; indeed such a case has been described by Moos (*Zeitschr. f. Ohrenh.*, Bd. xii., s. 96), and if the theory that the basal part of the cochlea is apportioned for the perception of high notes be correct, then an affection of that portion of the organ of Corti near to the fenestra rotunda would obviously produce the loss of hearing power for these notes. Furthermore, it has been suggested that from its

anatomical position that portion of the organ of Corti near the fenestra rotunda and the vestibule is more liable to disease than the more secluded parts. The suggestion appears to be a very reasonable one, but a larger accumulation of pathological evidence of the nature above described, is required before this explanation can be definitely accepted.

It will naturally be understood that an affection of the auditory nerve either in its trunk or in its projection in the brain may bring about a loss of hearing power for notes in different parts of the scale according to the fibres involved. Hence the inability to hear high notes only refers, so far as differential diagnosis is concerned, to affections of the labyrinth, and even in these cases it must not be relied on too implicitly.

The means by which a loss of hearing for the low notes is brought about is a matter of much more importance. And this is so for several reasons. In the first place, as observed above, testing the hearing power for the low notes is more reliable as a means of diagnosis; but what is of still more importance is the fact that the human voice as used in conversation is pitched in the lower portions of the audible musical scale. It follows, therefore, that any means devised for the relief of deafness must be directed more particularly to the restitution of the hearing power for the low notes.

In view of these facts it is remarkable that no attempt has been made to explain the great loss of hearing for the low notes in cases of disease of the middle ear, the condition in which these notes are lost to the patient.

The following explanation appears to me to account for this loss.

The loudness of a sound is, within wide limits, and certainly in the cases under consideration, proportionate to the energy transmitted to the fluid in the labyrinth. Now, since the mass of the ossicles is constant, the energy transmitted is proportional to the square of the velocity with which the ossicles move. Further, the velocity is proportional to the frequency and to the amplitude of the vibrations which the ossicles undergo. When, therefore, the frequency becomes

diminished by lowering the pitch, the amplitude of the vibrations must be increased in order to obtain the same amount of energy. Now, it is just in cases of middle-ear disease that the amplitude of the vibrations is restricted; hence, in this class of diseases the hearing power is much more seriously affected for the low notes than for the high ones.

Note.—The method of preparing the cochlea referred to is modified from one which I obtained from Böhm, and is as follows.

After removing the whole petrous portion of the temporal bone as soon after death as is admissible, file through the convexity of the superior semicircular canal, pierce the fenestra ovalis, and put the mass into a large quantity of a 4 per cent. solution of formalin. After it has been in the formalin solution for forty-eight to seventy-two hours put it into a large quantity of Müller's fluid. Böhm omits the previous fixation with formalin, but, on account of the rapidly penetrating power of this agent, I think it is of great advantage, particularly as a certain amount of disintegration among the cells must have already occurred.

The preparation remains three weeks in Müller's fluid at the temperature of the room, the fluid being frequently changed. At the end of the three weeks the vessel containing it is put into a stove kept at a temperature of 33° C., the Müller's fluid being changed frequently as before. This treatment in the stove lasts other three weeks.

The preparation is then cut down with a fretsaw to the smallest dimensions possible without injuring the structures, and a small hole is filed in the cochlea. It is then washed out for forty-eight hours in running water, and placed in 70 per cent. alcohol, where it is kept for three days in the dark.

It is now ready for decalcification, which takes place in a solution of five parts of strong, fuming nitric acid to 100 parts of 70 or 80 per cent. alcohol. The solution is to be changed twice a week, and the preparation must be kept in the dark. Decalcification is complete in from four to six weeks, at the end of which the preparation is put into a large quantity of

70 per cent. alcohol to extract the acid. It is then put in 90 per cent. alcohol for another fortnight, then for three days into 96 per cent. alcohol, and for two days into absolute alcohol, and is then embedded in celloidin.

The embedding must be very carefully carried out, beginning with a very thin solution for a fortnight, then with a solution of medium thickness for about a fortnight or three weeks, and then with a thick solution for a week.

The orientation must be such that some of the sections show the modiolus from apex to base. In examining the sections particular attention should be directed to the nerve-fibres in the lamina spiralis ossea and to the ganglion spirale.

ANGINA ULCEROSA BENIGNA.

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ANGINA ulcerosa benigna was first described in 1890 by Heryng, who had seen nine cases in nine years. The affection is characterized by the presence of a solitary superficial ulcer or excoriation on the upper part of one of the anterior faucial pillars. The ulcer is oval, measuring about 1 cm. in its long diameter, shallow, with sharp edges, of grey colour, and without inflammatory zone. The surrounding mucous membrane may appear normal, or may be slightly red and swollen. The ulcer heals in from ten to twelve days, and leaves no cicatrix.

Less frequently the ulceration is bilateral, as in one of Heryng's cases, and in two reported by Sendziak: the ulcers in one of the latter were seated on the posterior pillars. In two of Rosenberg's cases, in addition to the ulcer on the anterior faucial pillar, a similar one was present on the tonsil of the same side.

The patient usually complains of painful deglutition, which is out of proportion to the signs of local inflammation. There is slight general disturbance, which as a rule passes off quickly; the pain on swallowing, however, continues until the ulcer begins to heal. The affection is sometimes ushered in with symptoms of an ordinary angina tonsillaris, as in three of Heryng's cases in which the ulcer formed after the fever had subsided.

In none of the cases reported has a connection with syphilis, tuberculosis, or other constitutional disease been

proved to exist. Freudenthal refers to five cases in which an ulcer closely resembling that described by Heryng was associated with rheumatism; in four of these, however, the ulcer was seated on the posterior wall of the pharynx, while in the fifth it was on one of the posterior faucial pillars; the affection, therefore, was probably not identical with angina ulcerosa benigna. M. Schmidt mentions a case in which one of Heryng's benign ulcers appeared in the course of an attack of diphtheria. Sendziak assigns as the cause in one of his patients the abuse of tobacco.

Heryng examined the coating of the ulcers and found that it consisted of dead epithelial cells between which were two forms of streptococci (*S. monomorphus* and *S. variegatus*). The subsequent investigations of Lubliner, Masucci, and Sendziak have not fully confirmed the etiological relation of these micro-organisms to the ulcers.

The treatment ordinarily employed in acute inflammation of the fauces suffices.

The following cases of this affection have been observed by the writer:

Case 1.—The patient was a healthy man, about 28 years of age, and was under treatment for nasal obstruction. At one of his visits he complained of sore throat. As he was a professional singer and inclined to pay undue attention to any trivial ailment in this region, and as the only apparent disturbance was slight redness of the fauces no special treatment was recommended. Three days later, however, he returned complaining still more of painful deglutition. An ulcer was now found on the lower half of the left anterior faucial pillar, the neighbouring parts appearing normal. The ulcer was superficial, greyish, circular, and about half a centimetre in diameter. Syphilis at once suggested itself, but after careful investigation it was felt that it could be excluded with certainty. Besides, the pain complained of was much more severe than that accompanying a small mucous patch. The ulcer was painted at intervals with a weak caustic solution; the pain gradually diminished; and the region assumed its normal aspect within a fortnight. The patient has been seen

occasionally since the above attack, but there has been no recurrence.

Case 2.—Mr. L., aged 32, had consulted me on account of the extrusion of cheesy masses from the left tonsil. At one visit, after the tonsil had been reduced and was healed, the stump was painted with a solution of iodine. Four days later he returned complaining that the throat had been sore during the interval, and that on the day following his last visit he had noticed what seemed like a small burn on the left side. On examining his pharynx a superficial, circular, grey ulcer was found which could be covered by the head of a tack, situated on the left anterior faucial pillar close to its edge; the surrounding mucous membrane was normal. A gargle only was prescribed. In about a fortnight after the onset the ulcer disappeared.

Case 3.—Fred —, aged 15, complained of sore throat of three days' duration. During the previous three years he had had frequent similar attacks.

On examining his throat, he was found to be suffering from acute lacunar tonsillitis. The faucial tonsils were moderately enlarged and presented areas of exudation; a small patch was noted on the left lateral wall of the pharynx; and on the pharyngeal tonsil and lingual tonsil small white points were also observed. Chlorate of potash and iron internally, and an astringent gargle were prescribed.

Four days later he returned. In the interval the throat had been very sore, especially on the left side, but on the preceding day the pain had begun to subside.

Four superficial ulcers were now found on the lower part of the left anterior faucial pillar: two were united at their peripheries and two were isolated. The diameters of these measured 2-3 mm. They were clean, greyish, not elevated, and with no inflammatory areola.

On the following day the ulcers were smaller and more superficial, and there was no pain. Two days later only the faintest traces remained.

Case 4.—Mr. G., aged 31, who was being treated for nasal polypus, complained at one of his visits of sore throat.

Five days previously he had bicycled into the country for the first time. Owing to the unusual exercise, and to hay fever from which he was suffering, he felt out of sorts afterwards. Two days later he had a severe "bilious attack," and the throat became sore. The pain continued throughout the three days preceding his visit, but did not prevent him from attending to business.

On examining his throat two small ulcers were found on each side. These were extraordinarily symmetrical both as to shape and site; the following description therefore applies equally to both sides. One ulcer was seated on the anterior faucial pillar about its middle and ran along its edge. It was 6-7 mm. long, and about 2 mm. in breadth. The other ulcer was oval, measured 2-3 mm. in diameter, and was seated on the tonsil. While the parts were in repose the peripheries of the two ulcers were in contact; on drawing forward the anterior pillar, however, a narrow strip of healthy tissue was found to intervene. Both ulcers were quite superficial, and had light grey clean surfaces; they differed only in shape. There was no inflammatory areola.

Two days later the appearances were almost unchanged.

Eight days after his first visit, the ulcers on the tonsils were merely indicated by minute white specks; those on the anterior faucial pillars presented a slight loss of tissue, which on one side took the form of two tiny notches.

Case 5.—Robert M., aged 23, complained of sore throat of two days' duration. He had had no previous attack. On examining his pharynx the small oval ulcer characteristic of angina ulcerosa benigna was found on the left anterior faucial pillar close to its junction with the tongue.

In the first two cases above reported the ulcer was solitary, and corresponded in appearance and site with Heryng's description of angina ulcerosa benigna; they may therefore be regarded as typical examples of this affection. Doubts may be raised, however, as to the propriety of including Cases 3 and 4 in this group. The former was atypical in its association with acute tonsillitis and in the multiplicity

of ulcers; the latter, in that the ulcers were multiple and bilateral.

The five cases have been grouped together because in all of them the ulcers were identical in appearance—leaving out of account some insignificant variations in size and shape. Further, they were all situated on the anterior faucial pillar, except in Case 4, in which a second ulcer was contiguous on each tonsil. The pathological changes therefore leading to the formation of these ulcers were probably much the same in all four cases.

It is difficult to form an idea of the mode of origin of the solitary ulcer in a typical case of angina ulcerosa benigna, there being usually but little in the history or local appearances to give a clue. On the other hand, when a case presents itself with ulcers on both sides of the fauces which are perfectly symmetrical in shape and arrangement, it is evident that they are due to a constitutional disturbance, which most probably is nervous in character.

In short, we would regard these ulcers as of herpetic origin. Such superficial lesions—they are little more than excoriations—are just what might be expected after rupture of good sized vesicles. Too much importance must not be attached to the fact that a vesicular stage has not been observed. Herpetic vesicles in the mouth are very short lived; and in cases of recurrent herpes which have been watched for long they have been seen only on very rare occasions.

Angina herpetica is quite distinct from the affection under consideration.

The site of these ulcers, on the anterior pillar of the fauces close to its edge, is specially exposed and liable to injury. In Case 2 the ulcer appears to have resulted from painting the region with a moderately strong solution of iodine; it may also be mentioned that this patient was highly neurotic. An acute tonsillitis was the exciting cause in Case 3. In Case 4 the probable cause was a chill in one suffering from hay fever, whose nervous resistance was lowered.

The medical practitioner is not likely to be consulted regarding angina ulcerosa benigna, owing to its comparative

mildness and short duration. Four of the cases above reported were met with accidentally while the patients were under treatment for other ailments.

An acquaintance with the clinical aspect of angina ulcerosa benigna is desirable, owing to the readiness with which it might be mistaken for secondary syphilis. On careful examination, however, the difference between the ulcer and a mucous patch would become apparent, and a consideration of the duration, severity, and concomitant symptoms would lead to a correct diagnosis.

LITERATURE.

- Heryng, T.—“Ueber benigne Pharynxgeschwüre,” *Verhandl. des X. internat. med. Congr.* Bd. iv., 12. Abth., p. 74.
- Masucci.—“Ulcerations pharyngées de Heryng,” *Annales des maladies de l'oreille*, etc., Dec. 1891, p. 811.
- Sendziak, J.—Ein Fall der sogenannten “Angina ulcerosa benigna” (Heryng), *Monatsschr. f. Ohrenheilk*, July 1892, p. 199.
- Sendziak, J.—A second case of so-called “Angina ulcerosa benigna” (Heryng), *Journ. of Laryngol. Rhinol. and Otol.*, Aug. 1892.
- Rosenberg, A.—Gutartiges Pharynxgeschwür. *Die Krankh. der Mundhöhle, des Rachens und des Kehlkopfes*, Berlin, 1893, p. 109.
- Freudenthal.—“On rheumatic and allied affections of the pharynx, larynx, and nose,” *New York Medical Record*, 16th Feb. 1895, p. 198.
- Schmidt, M.—*Die Krankheiten der oberen Luftwege*, 2te Aufl., 1897, p. 530.

THE OPERATIVE TREATMENT OF UMBILICAL HERNIA.

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DURING the past four years it has fallen to my lot to examine in hospital and in private practice a large number of cases of umbilical hernia.

I propose to arrange them, in respect of operative treatment, into three groups: (a) Operable, (b) Doubtful, (c) Inoperable. A brief statement of the leading features of each of these varieties is necessary in order to make the meaning of the terms clearly understood.

(a) *Operable*.—The hernia does not exceed in size a hen's egg. The bowel is easily reduced, and the amount of omentum adherent to the sac is not large. The opening admits of one or two fingers, and its margin is well defined.

The patient should be in good health, and not over 40 years of age. Two other favourable features may be mentioned; first, that the hernia has not been in existence for more than two years, and second, that the patient is not putting on fat rapidly.

(b) *Doubtful*.—The hernia varies in size from that of an orange to that of a foetal head. The contents are made up of a considerable quantity of omentum which occupies a number of pouches in the wall of the sac, and to which it is usually firmly adherent. The opening admits of three or more fingers in addition to the pedicle of the omentum. The recti are pushed apart, and when the erect attitude is assumed the hernia

increases, becomes larger in size, and ill-defined at its base. The patient is approaching 50, is stout, and experiences considerable discomfort when obliged to lie on her back for more than a few hours. A cough, erythema intertrigo, or haemorrhoids, common in these cases, may interfere with healing and permanent good result from an operation.

(c) *Inoperable*.—The patient is about or over 50, is very stout, has a large pendulous abdomen, and attributes the great increase in the size of the hernia to attacks of sickness with vomiting, to the strain of coughing, or to her stoutness. All the anatomical changes mentioned in connection with the doubtful cases are here much exaggerated, and it would seem as if the abdomen were too small to receive the contents of the sac even after excision of the omental part of the hernia. Approximation of the recti would be impossible, and closure of the ring would be effected with extreme difficulty, and would involve so much strain upon the ligatures that in all probability they would yield and be absorbed before perfect union had taken place.

In respect of the relative frequency of the different groups, the operable together with the most promising of the doubtful series were in a minority; the inoperable formed a large majority.

Nearly all the patients had at one time or another worn a truss or some form of abdominal support, and the universal opinion was decidedly unfavourable to that method of treatment.

The results of the modern treatment of inguinal and femoral hernia prove quite conclusively that a cure is effected in the majority of cases, and the fact is so well recognized and admitted that it seems unnecessary to adduce statistics. It is, however, important to remember that a permanent good result depends largely, though not solely, upon the restoration of the parts to their normal position.

This is easily accomplished in inguinal hernia, as operative treatment is now resorted to at an earlier stage, before the valvular character of the canal is lost and the natural oblique passage is changed into a direct opening.

The frequent relapses in the early history of the radical operation were due to difficulties in rendering the support efficient when a direct opening was concerned, and the fact holds good at the present time and is thoroughly appreciated by surgeons in the treatment of femoral hernia. It remains to be said, however, that even in the latter form of hernia the bowel can be prevented leaving the abdomen, and a fair guarantee of success given if the crural ring is not unduly large. The author has a number of cases under observation operated on over five years ago which have shown so far no signs of relapse.

The treatment of umbilical hernia by operation has not received from surgeons and teachers the attention the importance of the subject deserves. It has been the practice to gloat over the size of the hernia rather than to point out to the student the advantage of early operation and the conditions upon which success depends. It must, however, be conceded that many of the procedures for the closure of the umbilical opening have not met with much favour; and, further, it must be admitted that surgical literature does not proclaim many cures after surgical interference.

I append the history of my first case operated on four years ago, and, as the patient has remained perfectly well and is at the present time in the enjoyment of excellent health, I am disposed to consider that the measures I then adopted and have since practised in other cases have withstood the test of time, and have proved quite effectual. I would again impress upon those who come across cases of umbilical hernia the fact that success is largely bound up with anatomical factors, and that these can only be controlled in the early development of the hernia.

CASE.

Mrs. S., aged 52 years, was seen in consultation with Dr. Macmillan, Pollokshields, in October, 1895.

At the time of my visit she was recovering from a severe

attack of pain and vomiting, brought on by a mass of omentum and bowel being retained in the sac. She had had a number of such attacks, but this she declared was the worst. Her condition had given rise to some alarm on a previous occasion, and at the consultation which was then held her medical attendant suggested that operative measures should be taken to prevent a recurrence of the attacks, but the surgeon gave no hopes that operative interference would gain that end. The case appeared to me to be a favourable one for operation. The patient was in excellent health, and promised to be a good subject in every way. The hernia was about the size of a Tangerine orange, contained adherent omentum but no bowel. When the bowel slipped into it it became much larger. The opening admitted of two fingers, which was not large considering the hernia had been in existence for some years.

OPERATION.—Preparation of Patient.—Careful dieting and attention to the state of the bowels are important means of preventing sickness and much abdominal discomfort. If the patient can be persuaded to be in bed for a week or ten days, to accustom herself to the dorsal position and to the use of the bed pan, it will be found to greatly simplify the after-treatment.

Details of Operation.—A vertical median incision is made, and the skin and sac are divided the entire length of the tumour. Adherent omentum is detached and returned into the abdomen, or removed as may seem best to the operator.

The sac is separated from the skin and fat down to the outer margin of the ring, any adherent points being divided with scissors. The forefinger of the left hand is introduced within the ring to guide the knife, which is made to divide the fascia continuous with the sheath of the rectus at the point where it becomes blended with the peritoneum of the sac.

Latterly I have used a pair of fine, blunt-pointed scissors to divide the fascia, thus minimizing the danger of puncturing the sac and of injuring its vessels.

The separation of the peritoneum within the ring must be conducted with great care, as all the structures at this point

are pretty firmly joined together. It is best accomplished by pulling the rectus aside and dividing any unseparable adhesion with scissors or knife. Beyond the ring the peritoneum separates readily, and it should be stripped from the sheath of the rectus for about three-quarters of an inch to provide room for the body of the sac.

The sac is now returned into the abdomen, and fixed in position by two lateral or median ligatures—one passing through the structures at the upper angle, and the other at the lower. The recti are now more thoroughly exposed and brought together by stout cat-gut sutures. The sutures pick up in the first instance the muscle on one side, and the ends are carried beneath the one on the opposite side and brought out at a point three-quarters of an inch in from the free edge. This slight overlapping of the muscles efficiently supports the sac, and supplies the best protective barrier to the hernia. Silk-worm sutures are passed through skin, fat, fascia, and superficial parts of recti, to obliterate any trace of a cavity between these structures and to avoid the necessity for any form of drainage. A continuous suture of cat-gut is now passed through the fascia at the margins of the ring, and the superficial sutures are then finally tied.

A broad elastic bandage keeps the dressing in place, and supports the abdomen generally. After six or eight weeks in bed, the patient is allowed to move about as usual, wearing a light abdominal belt.

ANTISTREPTOCOCCUS SERUM: ITS APPLICATION IN THE TREATMENT OF SCARLATINA ANGINOSA.

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WITHIN recent years, owing to the advance made in our knowledge of micro-organisms and the products of their vital activity—due to the perfecting of the methods of bacteriological research and investigation—a new phase of medical treatment has been inaugurated, viz. serumtherapy. This mode of treatment is applicable, in the widest sense, to all those diseases which have a microbic origin, and in which a serum, either antitoxic or antimicrobial in nature, can be obtained from an animal which has been rendered immune, by various methods, to the disease-producing micro-organism. These methods are manifold, but their ultimate result is the same, *i.e.* the formation of a serum which has an inimical influence either on the microbes or on their toxins. The results which have attended the use of antidiphtheritic serum have encouraged bacteriologists to pursue their investigations of the biology of other organisms, and a number of sera are now manufactured which are claimed by their respective discoverers to have certain specific effects. Examples of these are found in the sera of tetanus, cholera, bubonic plague, yellow fever, and streptococcal infections. The beneficial effect of antidiphtheritic serum is admitted by most observers, but sufficient trial has not been given to the other sera mentioned to warrant a definite expression of opinion. Encouraged by the good results in a small number of cases treated by these

sera, men have shown a tendency to form hasty generalizations and to assume an attitude which is not justified by the number of facts collected. More especially is this the case in regard to antistreptococcus serum, much harm having been done and the issues having been confused by failure to employ the serum in a scientific manner. This has also been felt in cases of diphtheria combined with a streptococcal infection, where, in spite of manifest improvement in the local condition of the throat from the use of antidiphtheritic serum, the patient has succumbed to streptococcal intoxication, and the serum has been credited with failure. The application of each serum should be directed against a particular organism, and just in proportion as the infection is a mixed one, so we should expect, unless a compound serum is used, to find our results unsatisfactory.

While from the laboratory point of view it would be difficult to give precedence to the study of any one micro-organism and its toxine, from the purely clinical standpoint the study of the biological characteristics of the streptococcus will always hold a foremost place. It is probably the most widely distributed form of microbic life; it is more variable in its morphological development than any of the others; it is unequal in the matter of biological characteristics; and it is the most far-reaching in its pathogenic effects.

The difficulty of attributing any definite pathology to the streptococcus and its association in so many dissimilar diseased conditions, such as erysipelas, puerperal fever, septic wounds, diphtheria and scarlet fever, readily accounts for the wide diversity of opinion that exists as to its nature and effects.

Until quite recently, bacteriologists had taken the view that in all the above mentioned diseases there was a distinct species of streptococcus concerned. Indeed, as many as twelve different species of streptococci have been described. Especially was a sharp line of distinction drawn between the streptococcus erysipclatis of Fehleisen, described by that investigator in 1883, and the streptococcus pyogenes of Rosenbach, described in 1884.

Within the last five years, however, these views have been abandoned, and Continental, American, and British observers,

with the one notable exception of Klein in this country, are now almost unanimous in their belief in the unity of the streptococcal germ, so far at least as toxine formation is concerned.

The differences in size, form, and effects of cultures of streptococci may be accounted for by the environment of the organism. These characters may vary according as the germ is cultivated on natural or on artificial media, or even on different natural or artificial media, according to their origin. Thus it was found in the cultivation of the streptococcus, that in some instances the loops were long and twisted, with an extremely high degree of virulence; in others they were short and straight and comparatively innocuous in their effects. This led to the belief that the long streptococci were always very virulent, and the short chains harmless. Marmorek, however, by injecting the same culture into rabbits and mice found that in subcultures from the blood of the former animal the chains were longer, and from the blood of the latter were shorter, although the virulence remained the same in both. By inoculating other animals, the same facts were brought out. As another proof of his contention, he offers the further experimental fact that streptococci from various sources and of varying grades of virulence may, by successive cultivation in the bodies of animals, all be brought to a point of virulence identical, all producing in animals an acute septicaemia. In a report of his work in this line, published in the *Annales de l'Institut Pasteur*, July, 1895, he sums up in these words: "Nos expériences confirment l'opinion de ceux qui regardent toutes les affections streptococciques de l'homme comme dues à un microbe unique."

Petruschky has collected a number of streptococcal affections of man, and submitted them to accurate bacteriological examination. He concluded that various streptococcal conditions pass one into the other or proceed from one another. This observer adduces the case of an infant who suffered from a typical attack of erysipelas from a scratch in the nasolabial fold, while the mother suffered from mastitis, which, however, showed no erysipelatos character.

Why a streptococcus should produce in one case an abscess, in another case erysipelas, and in a third pyaemia, is probably attributable partly to differences in virulence and in resistance-capacity, and partly to the seat and mode of entrance into the body.

The general recognition of the unity of the streptococcus microbe warranted the belief that the first step had been reached in the preparation of an antitoxin. The chief difficulty consisted in the inability to produce a potent filtered streptococcus toxine; for, while there is good reason to believe that some soluble poison of a very powerful nature is produced in the bodies of infected persons, yet the filtrates of cultures of streptococci have but little toxic action. Recently Roger has succeeded in immunizing rabbits by injecting filtered cultures heated to 120° C., but no attempt has yet been made to produce an antitoxin.

Having settled in his own mind the question of the unity of the germ, Marmorek next proceeded to endeavour to prepare a serum which would destroy it. Roger, Behring, Mironoff, and Lingelsheim had succeeded in establishing many points of interest in connection with the development and effects of the germ itself, and had even prepared a serum that was partially successful in destroying it. However, these observers never succeeded in preparing a serum of sufficient potency to act effectively upon the severer forms of the infection.

It has been clearly demonstrated that "laboratory sera," such as were formerly made by employing a selection of members of the rodent family, are so attenuated as to be almost inert. Such animals do not seem to be able to resist the toxic effect of repeated injections, and hence the serum procured from them is not of sufficient potency to act with much, if any, effect on the germ. This seems to have been the difficulty with the sera made by the four observers above mentioned. Marmorek, recognizing this difficulty, suggested that larger animals should be used in the preparation of the serum. This observer first used the sheep, then the ass, and latterly the horse. The horse tolerates large doses of the unfiltered toxine remarkably

well and yields by far the largest quantity of serum of any animal yet tried. Marmorek considered that in the preparation of the serum it was necessary (1) to exercise good judgment in the selection of animals, (2) to properly care for them during their treatment, and (3) especially to avoid a too early withdrawal of the blood.

In the preparation of antistreptococcus serum, as is the case in the preparation of products of like character, time, skill, and experience are required, and sera manufactured carelessly, and with a lack of one or more of these factors, have been put on the market and have led to failure and disappointment in their use.

The process of manufacture is necessarily a slow one, and a horse requires to be under treatment at least a year before the serum reaches its maximum antistreptococcic strength. The blood of the animal must not be withdrawn till some time after the last injection has been made, as during the reaction the blood is actually toxic, and, if injected into rabbits during this time, will invariably kill them in from five to ten days. At the British Institute of Preventive Medicine Horse Farm at Sudbury the *modus operandi* is as follows, viz.: Fifty cubic centimetres of a serum bouillon culture of (1) a streptococcus derived from an acute abscess, and (2) a streptococcus from a fatal case of acute septicaemia, are usually injected. Previous to 1898 the serum prepared was "univalent." The streptococcus of erysipelas was employed, its virulence being enhanced by Pasteur's method of passage through rabbits. All the cases of scarlatina anginosa in the subsequent series were treated with a serum prepared in this manner.

After the injection of the culture the horse's temperature rises steadily till in six or seven hours 41° centigrade (105.4° Fahr.) is registered, 38° centigrade being the normal temperature of that animal. A decline then sets in and the temperature is usually normal the following day. At various periods samples of blood are drawn to estimate the preventive and curative power of the serum and also to determine the length of time during which the serum remains toxic after inoculation.

According to experiments made at the British Institute of

Public Health, the serum does not remain toxic for so long a period as that found by Marmorek in his investigations. A month is allowed to elapse between the last injection and the withdrawal of the blood from the horse.

The serum is preserved by being kept cold, and no antiseptics are added for this purpose. Roux, who has had a large experience in the manufacture of antitoxins and sera, asserts that antiseptics can do no possible good and are capable of doing a great deal of harm. The sera, in his opinion, are not preserved by antiseptics, as is popularly supposed; they will only keep a certain time; they must be kept at a proper temperature in order to retain their vitality, and, if so kept, will remain potent as long without antiseptics as with them.

Up till recently, and long after the author began to use the antistreptococcus serum in anginous scarlet cases, the question of the unity of the streptococcal germ was accepted in all its entirety, whether viewed from the morphological or from the biological aspect. Lately, however, certain experiments have been made by observers whose results are so unanimous and cannot be contradicted that the question in one of its aspects at least is reopened. These experiments have shown that whereas a serum of a horse immunized against a certain streptococcus will protect the animal against the streptococcus used to immunize it, there is no protection conferred on the animal against a streptococcus derived from a different pathological condition. Although the ultimate identity of streptococci from various sources cannot be doubted, yet there seems to exist a subtle distinction in the bactericidal substances which streptococci, associated with different pathological manifestations in the human economy, produce in the blood of animals into which they are injected.

The most interesting experiments in this line were made by Van de Velde. This investigator selected two streptococci, *A* and *P*, each of which when injected into a rabbit's ear produced erysipelas without any streptococci being found in the blood and internal organs. With each of these microbes he immunized a horse and obtained two sera. The serum obtained from horse *A* protected against streptococcus *A*, but

had a very feeble protecting influence against streptococcus *P*. The serum obtained from horse *P* protected against streptococcus *P*, but failed to protect against streptococcus *A*. This experimenter also went one step further. Having injected five cubic centimetres of one of these sera (*A* and *P*) into each of two rabbits, he inoculated the right and left ears of these animals with streptococcus *A* and *P* respectively. In the rabbit injected with serum *A*, only the ear inoculated with streptococcus *P* developed erysipelas, while the converse occurred in the case of the other animal. The position which this observer takes up was still further strengthened by the fact that he immunized a horse with both streptococci *A* and *P*, and obtained a serum which would protect against both these organisms. From these results he recommends that animals employed to yield serum should be injected with a number of streptococci from various sources in the hope of obtaining a serum active in its bactericidal properties against as many streptococci as possible.

This observer also investigated the agglutinating power of his sera, and he finds that it is always in direct proportion to the power of protecting. Thus, in his experiments quoted above, he found serum *A* agglutinated streptococcus *A*, and serum *P* agglutinated streptococcus *P*, and that serum *A* + *P* agglutinated both.

The outcome of these experiments was the suggestion that the patient's blood should be tested against a number of streptococci to get some idea of its agglutinating power. These streptococci could have corresponding sera. In this way, having found the streptococcus which was best agglutinated by the patient's blood, the serum could be selected which would probably have the most beneficial effect in stemming the progress of the disease.

The practical conclusion arrived at by this observer was that each variety of streptococcus was able to produce in the animal, into which it was injected, a particular antimicrobial body which was inimical to the life of that streptococcus. This statement is not an exclusive one, and an antimicrobial serum, which did not correspond to a particular variety of

streptococcus, might have an influence on that organism, although not to such an extent as on the corresponding streptococcus.

These facts open up a very important aspect of the case in regard to the practical application of the serum in human disease.

The difficulty of preparing a potent germ-free toxine of a streptococcus has been adverted to previously. All experiments in this line have therefore been conducted with an antimicrobial, and not with an antitoxic serum. The author was much impressed by the want of uniformity which attended the use of the serum even in cases which seemed in all respects comparable to one another. The argument would therefore be urged that in scarlatina anginosa there may be different varieties of streptococci present so far as their vulnerability to the bactericidal substances contained in the serum is concerned, and that, according to this, did improvement take place or not.

Although different races of streptococci require a corresponding bactericidal serum, and are affected to a modified degree only by other sera, yet the toxines produced by the various streptococci may be essentially of the same nature, and it seems reasonable to hope that they may be counteracted by a single antitoxin.

The researches of Calmette lend support to these suggestions; for he has shown that the poisons of the most various serpents and also of the scorpion, while they differ somewhat in their action upon animals, nevertheless are counteracted by one antivenin.

Bearing these considerations in mind, it seems that our aim should be to produce, if possible, an antitoxic rather than an antimicrobial serum for the treatment of diseases caused by streptococci.

That there is good reason to believe that the bactericidal substances formed in the bodies of animals into which different forms of streptococci are injected, are varied, is proved by Renon's observations in cases of scarlet fever. This investigator obtained a streptococcus from the blood of a scarlet

fever patient. Tested on mice and rabbits successfully inoculated by this infection, it was absolutely unaffected by Marmorek's serum. The same serum, however, checked the growth of a streptococcus obtained from Marmorek, and this streptococcus was a more virulent one than that from the scarlet fever case, so that it could be inferred that streptococci of various kinds were found in man as well as in animals, some of which are subject to Marmorek's serum, and others are not. Mery also succeeded in isolating in cases of scarlet fever seven varieties of streptococci obtained from the throat, urine, blood, and glandular abscess. Six of these streptococci proved refractory to Marmorek's serum.

The specific materies morbi of scarlet fever has not yet been discovered. Some observers have described a parasite in the blood of patients, similar in some respects to the malarial parasite. Klein in this country has described a streptococcus which he has recovered from the blood, and also from the tubuli uriniferi of the kidneys. This observer has obtained the same organism from the teats of a number of cows supposed to be concerned in an outbreak of scarlet fever at Hendon. These opinions have not yet been confirmed, and are, in fact, strongly controverted by many competent observers in this country, so that the question must still be regarded as an open one.

Sternberg, in his *Text Book of Bacteriology*, 1897, says: "The specific infective agent in scarlet fever has not been demonstrated. In the diphtheritic exudate frequently seen in the angina of scarlet fever, a streptococcus is commonly found which appears to be identical with streptococcus pyogenes; and in the secondary infections which occur in the course of the disease or during convalescence when suppuration occurs, one or other of the common pyogenic micrococci is usually found, and is doubtless the cause of the local inflammatory process."

The question of the etiological factor in scarlet fever may long remain doubtful, but this, in the author's opinion, should not tie our hands in the treatment of the severer forms of the affection. All the severe complications, such as pseudo-mem-

branous angina, otitis media, endocarditis, and nephritis are due to the presence of streptococci.

Stripped of these complications, scarlet fever would not be a dangerous disease. These were the facts that justified in the author's opinion an extended trial of the new serum that was introduced for the treatment of this affection. (See Tables of Cases, pp. 260-268.)

Outline of Treatment.—The serum used was obtained from the British Institute of Public Health. It was kept in a cool place and was rejected if it showed any sediment. It was injected subcutaneously either into the loose cellular tissue of the abdomen or between the scapulae. The skin was first thoroughly cleansed with turpentine and methylated spirit, and then laved with a 1 in 40 solution of carbolic acid. The quantity injected at any one time never exceeded 20 cubic centimetres.

Four cases received only 10 cc.; fourteen cases 20 cc.; nine cases 30 cc.; four cases 40 cc.; one case 50 cc.; four cases 60 cc.; one case 65 cc.; three cases 70 cc.; three cases 80 cc.; one case 100 cc.; one case 110 cc.; one case 150 cc.; and one case 160 cc. In four cases only a single injection was made; in the remainder the condition seemed such as to warrant further injection. Simple measures were also taken to keep the mouth and nose as clean as possible. With this aim in view, boroglyceride or a weak solution of perchloride of mercury was used to keep the mouth clean, while boracic or sulphurous acid was used for the nose.

In addition to this, whisky and brandy were given according to the requirements of the case, while convalescence was established by a liberal use of iron and tonics.

Bacteriological Examination.—In all the cases a bacteriological examination of the throat was made, and in all streptococci were discovered, although in a varying degree. In all the cases, with the exception of two, there was a mixed infection. In thirty-four cases, cover-glass preparations made directly from the secretion of the throat, and subsequent streak and stab cultures, revealed the presence of streptococci and staphylococci. In eight cases three or more different micro-organisms were found; in three cases an almost pure

culture of staphylococcus pyogenes aureus was obtained; and in two cases an almost pure culture of streptococcus was demonstrated. Cover-glass preparations made from the three cases which subsequently, by means of cultures, proved to be staphylococcic infections, revealed one or two streptococci in the field. On the strength of this fact serum treatment was commenced. It was subsequently abandoned.

In six of the eight cases in which three or more organisms were found in the throat, streptococci, staphylococci, and diplococci were demonstrated; in one streptococci, staphylococci and a short rod, like the bacillus coli communis; and in another streptococci, staphylococci, diplococci, and a similar rod-like organism.

Incidence of Rashes after Injection of Serum.—Nine of the forty-seven cases treated were attended with rashes of varying description, and indefinite as to their time of occurrence. In addition to these rashes, there occurred in a number of cases a slight erythematous blush around the site of injection.

As regards their time of eruption, two appeared on the seventh day after the serum was injected; two on the eighth day; one on the tenth day; two on the eleventh day; one on the fourteenth day, and one on the fifteenth day.

In character these rashes varied from a punctate type resembling a scarlatinal eruption to a condition of the skin which suggested morbilli. A very common form was a simple erythema with periodic efflorescences of wheals. The rash was sometimes polymorphous. In one case an urticarial rash appeared on the eleventh day after the injection. On the following day it assumed the form of an erythema multiforme on the limbs, face, and scalp. In twenty-four hours thereafter it assumed the characters of an erythema on the trunk. This did not have the defined character it had on the limbs. It was not so much raised above the surface of the skin, its borders were not so defined, and it lacked the crimson colour it had twenty-four hours previously.

In the simple urticarial rash, the wheals were generally set on congested bases. In one case, however, there were no

congested bases, but a general blotchy rash with numerous wheals appeared on the seventh day. On the tenth day the wheals had entirely disappeared, and the rash which remained was of a measly type. In one case, on the tenth day after injection a morbilliform rash appeared on chest, back, and limbs. On the thirteenth day a deep coloured general erythema, not unlike a scarlet rash, appeared on the trunk. On the limbs it still remained measly looking.

As regards the duration of the rash, this varied from one to seven days. In one case a preliminary rash of wheals set on a measly base lasted for three days. It then quite disappeared for four days, when it again showed itself in similar characters to the first rash.

In some cases there was a coincident rise of temperature; in others there was none. The evening temperature in one case rose to 105.2° F.

In seeking an explanation of these cutaneous disturbances, it is obvious that they may be caused by the injection of the serum *per se*, independently of any adventitious element it may contain, or by some foreign elements. Frequently on the principle *post hoc ergo propter hoc* various manifestations have been attributed to the serum.

Three hypotheses are possible: (1) a special exciting action of the serum itself, (2) a personal susceptibility of the patient, or (3) a combination of these two conditions. As rashes of different characters are brought out in some individuals by the injection of normal serum, one is forced to the conclusion that this must be looked upon as a non-preventible accident of serum therapy, and that it will occur, in some measure at least, no matter with what care the serum is manufactured. There is no doubt, however, that with increased care in the preparation, together with an exalted "immunization value" of the serum, so that smaller quantities might be injected, these cutaneous disturbances will be reduced to a minimum. In the case where the evening temperature registered 105.2° F., the patient did not seem very ill, but had a high running pulse. The temperature and pulse rapidly subsided with the disappearance of the rash. In this case there was a somewhat

excitable disposition of the patient which probably accounted for the thermal disturbance.

Abscess at Site of Injection.—In spite of every precaution taken to prevent this, three abscesses formed at the site of injection of the serum, and in other three cases a certain amount of oedema supervened in the cellular tissue round about. In no case was the well-being of the patient interfered with.

Influence of Serum on Temperature.—The reduction of the temperature after injection of the serum has been insisted on by various observers, and considerable depressions accompanied by various critical phenomena, such as profuse sweatings, lowered pulse, and respiration have been noted. The author's experience does not support these views, but on the whole contradicts them. In twenty-one cases the temperature was taken half-hourly for some time after the injection was made, and although in the first register or two a lower reading may have been recorded, this was never sustained for any length of time. In all the cases treated the temperature was taken either two-hourly or four-hourly through the whole course of the illness, and the results were very variable. In twenty-seven cases there was no appreciable effect on the course of the febrile movement; in fifteen cases the effect was variable, sometimes showing a rise and at other times a fall; and in five cases the effect was such as to call for special comment. In none of the fifteen cases in which the effect was variable did the temperature ever show an upward or downward oscillation of more than 1° F.

Of the five cases which showed a marked result, the first showed a rise of 1.6° F. an hour after the serum was given, which was maintained for some time. In the second case the temperature at 6 a.m. was 105° ; at 1 p.m. 100.8° . The serum was injected at 1.45 p.m. The temperature registered at 3 p.m. 104.2° ; at 5 p.m. 104.2° , and at 9 p.m. 103.4° . In the third case there was a fall of 1.2° F. an hour after injection, which was not maintained; in the fourth case the temperature, fifteen minutes before the injection of the serum, registered 101° ; forty minutes after the injection was made it registered

104°; and in the fifth case the temperature registered 104° at 9 p.m.; serum was injected at 11.30 p.m., and the temperature at midnight registered 99.8°. In the last case, tepid sponging which had been used to depress the temperature was discontinued at 9 p.m. The temperature rose again to 103.6° in six hours. The pulse generally followed the temperature in all the cases; the respiration curve was very variable, and depended more or less on the condition of the lungs. These results have convinced the author that the serum is unreliable in its effects on the temperature curve.

Influence of Serum on Glandular Invasion and Suppuration.—Of the forty-seven cases under treatment, all had glandular affections to a more or less degree. In eight cases the condition was one of "slight bilateral cervical adenopathy." In twenty-eight cases the condition was one of "moderate bilateral cervical adenopathy"; in nine cases of "severe bilateral cervical adenopathy," and in two cases of "very severe bilateral cervical adenopathy." In none of the cases had surgical measures to be resorted to. The application of cold compresses was alone sufficient.

Of the two cases of very severe adenopathy, fluctuation was made out in one on the morning of the death of the patient. This was Case No. XXXIV., and was almost a pure staphylococcic infection. In the other case (No. XXIX.) no fluctuation could be made out but considerable oedema and brawny induration of the neck. In this case the submaxillary glands on both sides were much infiltrated, accompanied by well marked oedema of the face. The cellular tissue in the middle line of the neck was markedly oedematous, and this condition extended down as far as the level of the second rib. Under serum this condition greatly improved. Both the oedema and the induration almost entirely disappeared, and the infiltrated glands at the angles of the jaws could be distinctly delimited. No fluctuation could be made out. Infection was a mixed one, the staphylococci predominating.

Of the nine cases of severe adenopathy only one suppurated, and this case presented special features. This patient was admitted suffering from a mild attack of scarlet fever. On

the eighth day after admission she developed measles, and was transferred to a ward under the charge of a colleague. Under the agency of the combined virus of scarlatina and measles, the throat condition became much worse, ultimately resulting in a severe ulceration. With this condition a marked cervical glandular enlargement developed, for which poultices were applied. An abscess formed and pointed, and the pus was evacuated spontaneously. A drainage tube was inserted and the abscess cavity healed up. The pus formation in this case could scarcely be accounted for by the scarlatinal virus alone, as prior to the invasion of the measles the case was a mild infection. Cover-glass preparations made from the pus in this case showed an abundance of staphylococci with a few streptococci.

Of the twenty-eight cases of "moderate cervical adenopathy" and eight of "slight cervical adenopathy" none suppurated, although attended in several instances with extreme ulceration of the throat. In only one case therefore did suppuration occur at any stage of the illness. Bacteriological examination revealed in this case an almost pure staphylococcic infection.

This is an important fact when it is remembered that glandular infection with abscess formation is a common condition among children who suffer from scarlatina anginosa, more especially in the debilitated and strumous type of children treated in a large public institution.

Influence of Serum on Nasal Discharge.—In a large number of cases of scarlatina anginosa a nasal discharge is one of the earliest symptoms. The inflammatory process does not confine itself to the tonsils, anterior and posterior pillars of the fauces, uvula and pharynx, but also extends to the nasopharynx. There is good reason to believe that a nasal discharge is due to some ulcerative process in this region or in the upper part of the nasal mucous membrane. The factor which determines the *locale* of the commencing ulceration in scarlatina anginosa will always be doubtful, but, in cases where a nasal discharge is one of the earliest symptoms in the disease, a swab from the nasopharynx or an agar or gelatine tube sown from a

nasal discharge, after the nose has been thoroughly syringed with a weak antiseptic, almost invariably yields a pure culture of streptococci. The longer the delay in making a culture, there is less probability of a single organism being found. When an early nasal discharge is treated promptly with antistreptococcus serum, there is, in many cases, a considerable diminution, and in some a complete arrest of the discharge. The actual result depends on whether the serum is exhibited early or late in the disease. The effect of early injection where there is a copious nasal discharge is decidedly beneficial. If the treatment is delayed till the staphylococci outnumber other organisms in the cultures, the serum is useless in affecting the discharge.

The effect of the serum was shown particularly in the following four cases, which bear out the above remarks.

L. S., aet. $1\frac{1}{2}$ years. Admitted on fifth day of illness with sparse eruption, patchy and livid over the extremities. Throat covered with exudation. On removing this from the anterior pillars of the fauces and tonsils deep ulcers were observed. Profuse seropurulent discharge from both nostrils. Purulent discharge from both ears. Diarrhoea to the extent of five motions in the twenty-four hours. Moderate cervical adenopathy. 10 cc. serum injected on the morning after admission. On the following day a considerable diminution of the nasal discharge had taken place, and in character it was now a glairy mucus. In another twenty-four hours the discharge had quite ceased. Bacteriological examination: streptococci and staphylococci present, the former very abundant. An almost pure culture of streptococci obtained from nasal discharge.

W. D., aet. $5\frac{1}{2}$ years. Admitted third day of illness with a patchy scarlet rash. Profuse seropurulent nasal discharge. Moderate enlargement of glands at angle of jaw. Throat very dirty. Double otorrhoea. Nasal discharge ceased four days after admission. Had in all 30 cc. of serum. Bacteriological examination: streptococci and staphylococci both recovered from throat. Streptococci very abundant in nasal discharge.

E. B., aet. 2 years. Admitted third day of illness. Profuse seropurulent nasal discharge from both nostrils. Received 30 cc. of serum in all. Discharge entirely ceased twenty-four hours after the injection of first 10 cc. On the following day a slight re-establishment of the discharge took place, serous in nature. This ceased finally five days later. Bacteriological examination: streptococci very abundant. Staphylococci also present.

J. C., aet. 9. Admitted sixth day of illness. Profuse seropurulent nasal discharge. Serum injected, 10 cc. Nasal discharge ceased forty-eight hours after injection. Bacteriological examination: streptococci and staphylococci present. Streptococci extremely abundant in nasal discharge.

Influence of Serum on Ulcerative Conditions of Throat.—The beneficial effects which followed the use of the serum in some cases which showed extreme ulceration of the throat cannot, in the author's opinion, be disputed. Within twenty-four hours after injection the effect was in some cases very marked. In cases which showed a pseudo-membrane over both tonsils, there was often a successful exfoliation of the membrane, leaving a raw sore underneath, which, with the continued application of the serum, showed all the characters of a healthy granulating ulcer. In other cases where no pseudo-membrane was present, but which were characterized by deep unhealthy-looking ulcers, under serum these either spontaneously became much cleaner or nurse reported that the throat could be cleaned much more easily by the local measures used. In other cases where the serum was administered before ulceration commenced, it was observed that a sharp line of demarcation began to form at the outer edge of the necrotic patch, the exfoliation of which seemed to be hastened, leaving a healthy-looking ulcer underneath.

In some cases the local condition improved much more rapidly than the improvement in the general condition seemed to warrant, while in other cases a local improvement took place with no apparent change in the constitutional state.

In other cases, on the contrary, which seemed to be comparable in all respects, both from the bacteriological and

clinical standpoint, to those quoted above, no improvement, either local or constitutional, took place. The author has considerable difficulty in explaining these results. The treatment was commenced as early and pushed to the same extent as in the other class of cases. Moreover, in two cases where bacteriological examination revealed an almost pure streptococcic infection, and in which the treatment was pursued vigorously, no improvement took place, and both patients went rapidly downhill. It is a well-known fact that when two organisms live symbiotically the action of one is often enhanced by the mere presence of the other, and in many cases in a direct ratio to the preponderance of one over the other microbe. This theory may explain the varying results obtained in the cases of mixed infection treated in the series, but it does not throw any light on the unsuccessful issue of the two cases above mentioned, where the infection was practically a pure streptococcic one.

Influence of Serum on Albuminuria.—The incidence of albuminuria in scarlet fever is not confined to the anginous type, and is as commonly associated with very mild attacks of fever as with the more severe forms. This consideration makes it impossible to deduce much as regards the action of the serum in preventing or mitigating renal affections.

In three cases of those treated in the series nephritis supervened. In one case albumen, to the extent of a half, with blood appeared in the urine on the twenty-seventh day of illness, and was associated with general anasarca. In another case albumen with blood and associated with oedema of the legs and feet appeared on the twenty-fifth day of illness. In the third case a large quantity of blood and albumen, with organic debris in the sediment, appeared in the urine on the eighteenth day of illness.

Generally speaking the third week of illness is the period of incidence of renal affections in scarlet fever, so that these instances follow the usual rule.

Influence of Serum on Otorrhoea.—It is well nigh impossible to draw conclusions from anything that happens in ear discharges. These are known to be subject to considerable

variations when no treatment is exhibited. The thorough cleansing of the ear, also, with some simple antiseptic has often a good influence, either in arresting or lessening ear discharge, so that any value attached to the serum in influencing the flow is open to much criticism.

TABLE OF MORTALITY OF CASES.

Bronchopneumonia, - - - - -	5
Bronchopneumonia and severe diarrhoea, - - - - -	2
Extreme toxic infection, - - - - -	7
Uraemia with convulsions, - - - - -	2
Uraemia with diarrhoea, - - - - -	1
Extreme toxic infection and diarrhoea, - - - - -	1
Extreme diarrhoea with exhaustion, - - - - -	2
Diphtheria (post scarlatinal), - - - - -	1
Heart failure, - - - - -	2
Tubercular laryngitis and general tuberculosis, - - - - -	1
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The deaths attributed to diphtheria, heart failure, and tuberculosis could scarcely be due to inefficiency of the serum. In the case which succumbed to post scarlatinal diphtheria, the use of the serum was attended with the best results in arresting a very destructive and quickly spreading ulcerative process in the throat. The patient had a long convalescence. Fourteen days after she was allowed up, diphtheritic membrane appeared over both tonsils. Three days afterwards the patient became asphyxiated. Tracheotomy was performed but patient died two hours after the operation.

The two deaths due to heart failure occurred when both patients seemed quite out of danger so far as the toxic infection was concerned. The death from tubercular laryngitis and general tuberculosis occurred in a case mentioned previously. The patient was suffering from scarlatina on admission and developed a morbilliform rash, with coryza and lachrymation, on the eighth day of residence in hospital. Bronchopneumonia set in and apparently afforded a nidus for the tubercle bacillus.

If these four cases be deducted, the mortality is 20 or 42·5 per cent. of the cases treated.

The following abridged notes of four cases are taken from the ward journals. They are illustrative of four distinct types of the cases treated as regards both the local lesion and the general characters.

CASE 1.

Illustrative of Severe Implication of Throat.

M. B., $6\frac{1}{2}$ years. Admitted 1st April, 1898. Illness began last night with headache and sore throat. Vomited once. Rash seen to-day on trunk and limbs. Has had measles and whooping cough.

2nd April.—Throat is looking extremely dirty, and a foul excavated ulcer is present on the anterior pillar of the fauces on the left side. The right tonsil is raw and ulcerated towards the middle line of the mouth.

3rd April.—The throat this morning is very dirty, and there is considerable superficial necrosis of the soft tissues on the roof of the mouth.

4th April.—The throat is still looking dirty, and the ulcer on the left anterior pillar of the fauces is certainly extending. The back wall of the pharynx is superficially necrosed.

5th April.—10 cc. serum injected to-day. Throat is looking somewhat cleaner this morning.

7th April.—Ulcer on left anterior pillar of fauces is looking much healthier, and appears to be smaller to-day. A demarcating zone is observed to-day for the first time.

8th April.—Throat is greatly improved.

10th April.—Improvement continues, and all the ulcers, including those over the tongue, are healthy-looking.

13th April.—Granulations observed to-day over soft palate and posterior pharyngeal wall.

15th May.—Patient allowed up to-day. An examination of the throat shows considerable deformity. The anterior pillars of the fauces are drawn apart, exposing the two tonsils of unequal size, the right being smaller than the left as the result of its involvement in the ulcerative process. A very

small shred of the uvula is left. The soft tissues over the hard palate are puckered and glazed-looking.

CASE 2.

Illustrative of General Septic Absorption.

J. T., aet. $3\frac{1}{2}$ years. Admitted 3rd October, 1896. Illness began three days ago with sore throat, diarrhoea, and slight cough. Rash seen second day. No other infectious disease.

5.30 p.m.—Tongue, strawberry. Temp. $104\cdot4$, P. 140, R. 42. Rash fading on limbs, but well marked on trunk. Throat congested. Considerable exudation on both tonsils.

4th October.—Throat very dirty, especially on right side. Slight nasal discharge.

5th October.—Fairly quiet night. Less exudation on fauces.

6th October.—Superficial ulceration of the fauces, but little swelling.

7th October.—Small ulcer on uvula. Sloughy condition of left tonsil.

10th October.—There is a large slough on the left side of the soft palate to-day, and a similar necrotic area on the right side, but not so extensive. There are also one or two small ulcers on the uvula.

11th October.—10 cc. serum injected to-day.

15th October.—Condition of throat is improved. Ulcers looking healthier.

17th October.—Complains of pain at right elbow, especially on movement. There is some swelling around the joint. No redness. Tongue dry and glazed.

18th October.—Throat is still improving.

20th October.—Temperature is still elevated. Locally the throat is much improved. Elbow is very painful. There is still considerable swelling round the joint, and the forearm is held midway between pronation and supination. Movement causes pain.

22nd October.—Improvement in throat condition maintained. Temperature lower, pulse better in quality; slough

in throat is detaching itself, leaving a red healing surface exposed.

28th October.—This afternoon the elbow joint appears to be much more swollen, is very tender on pressure, and decidedly fluctuant. An exploring needle brought out a quantity of healthy-looking pus.

29th October.—To-day under chloroform made an incision on outer and posterior aspect of elbow, and evacuated several ounces of pus. Enlarged incision and explored with finger, and found elbow joint opened with sinuses passing up the arm towards the shoulder and round the posterior aspect of the humerus. No eroded bone found either in the joint or outside it. Drainage tube inserted. Soft palate cicatrizing.

2nd November.—A little pus exudes from the joint. Throat now perfectly healed.

3rd November.—Arm put up in a fixed position to-day.

20th November.—Sinuses practically healed. Cannot move the arm herself, and arm painful on passive movement.

7th January, 1897.—Patient dismissed to-day. The arm could be moved to a certain extent. In raising it, she had to support it by the left arm. Patient received in all 30 cc. of serum.

CASE 3.

Illustrative of a quickly spreading Bronchopneumonia, probably caused by Insufflation into the Trachea of small Necrotic Areas.

E. B., aet. 2 years. Admitted on second day of illness with sickness, vomiting, and sore throat. Rash seen to-day.

Throat is congested and extremely foul-looking over both tonsils. Slight nasal discharge.

30th March.—Fairly profuse nasal discharge from both nostrils, seropurulent in character. The throat looks dirty behind, and on the left tonsil there is a deep excavated ulcer. This is the only loss of tissue that can be observed. 10 cc. of serum injected to-day.

31st March.—Nasal discharge has ceased to-day from both

nostrils. Throat is looking much cleaner and the ulcer on the left tonsil is certainly not extending.

1st April.—Slight re-establishment of nasal discharge; 10 cc. serum injected.

3rd April.—Tissues over hard palate are necrotic; 10 cc. serum.

7th April.—Throat is not looking so favourable. The ulcer on the left tonsil is extending and also the area of necrosis over the hard palate; 10 cc. serum.

8th April.—General condition very unfavourable. A bright septic rash has appeared to-day over the elbows and ankles, extending on the right leg up to the knee.

10th April.—Patient is still very ill. Is taking her milk and stimulant well. Bronchopneumonia has developed. Both lungs affected generally.

11th April.—Patient passed a restless night. The throat is looking somewhat cleaner. Respirations this morning number 76. Condition is very unfavourable.

13th April.—Patient died at 5.15 this morning. Slight nervous twitchings and subsultus before death. At five o'clock the respirations reached 100. Patient received in all 50 cc. serum.

CASE 4.

Illustrative of Exhaustion from extreme Diarrhoea and attended with a fatal issue.

M. D., aet. $6\frac{1}{2}$ years. Admitted on second day of illness with sore throat, sickness, and diarrhoea. Temp. 102.8, P. 144, R. 40.

19th September.—Very restless last night. Rash generally distributed, especially well marked over arms, where it is somewhat patchy. Face pale with blotchy, hectic flush on cheeks. Patient heavy and ill-looking. Profuse watery discharge from nose. Pulse rapid and very shabby. Fauces, palate, and tonsils are extremely angry-looking, and both tonsils are covered with a purulent exudation. On the opposing surfaces

of both tonsils and on the uvula and anterior pillars of the fauces there are grey necrotic patches.

22nd September.—No change in local condition. Child is very restless. Three motions during the night. Nasal discharge as profuse. General condition is still extremely grave. Pulse suggests a failing ventricle contracting spasmodically and incompletely; 10 cc. serum injected.

24th September.—Pulse is distinctly better in quality to-day, less jerky and more sustained. The throat is improved and here and there are indications of granulations.

26th September.—A very decided improvement has taken place in condition of patient. The septic process in the throat is now quite localized, and the sloughs are separating. Nasal discharge has almost ceased.

28th September.—Improvement in throat condition is maintained. The sloughs have completely separated, and healthy ulcers are seen over anterior pillars of fauces and tonsils.

2nd October.—Patient has gone back since last note. Temperature and pulse have again risen. Pulse of spasmodic type it had at previous stage of illness. Diarrhoea began three days ago; four to five motions in the twenty-four hours.

3rd October.—Passed a very restless night. Throat is still looking well and the destructive process has ceased.

5th October.—Diarrhoea still continues; four to five motions in the twenty-four hours. The diarrhoea continued till the afternoon of the 14th October, when patient died suddenly. In the morning she was looking brighter. Throat was quite healed.

Remarks on the Treatment with a Consideration of Results.—The cases treated were patients in Belvidere Fever Hospital during the years 1896, 1897, and 1898, during which time the author was a Resident Assistant Physician.

As a premise it should be stated that the serum was put to a severe test, as all the cases were marked examples of the anginous type of scarlet fever. This was purposely done in order to determine the exact place antistreptococcus serum should hold in the therapeutics of this affection. In assigning

the proper value to a new remedy, unless its use is founded purely on an empiric basis, it is essential that it be exhibited upon rational and scientific lines. Especially is this the case with the application of sera to human disease. These are the fruit of work done for years along particular lines of study, and they cannot justifiably be administered to any case in a haphazard fashion. A bacteriological examination therefore should always be made. Antistreptococcus serum is directed against the ravages of the streptococcus on the human economy, and it is useless to expect good results from its employment in conditions where other micro-organisms predominate. The author would therefore put in a plea for exactitude in the use of sera. In the use also of antistreptococcus serum in cases of streptococcal invasion of surfaces exposed to the air, good results can scarcely be expected to take place if the treatment be delayed, as a primary simple infection is very soon converted into a mixed one. If the doctrine of the unity of the streptococcal germ, so far as toxine formation is concerned, be upheld, the formation of a powerful antitoxin would naturally be the best means of counteracting the ravages of any streptococcal affection. The difficulty of preparing a potent filtered toxine has been stated previously. Failing in their attempts in this direction, laboratory investigators set about making a bactericidal serum. It has been shown how a serum prepared from a specific streptococcus will only protect animals, in a restricted sense, either against the corresponding organism or one closely allied to it. Further, it has been proved that in proportion to the number of different races of streptococci injected into a horse, is the antimicrobial power of the resulting serum measured.

In the light of these facts there is a strong argument, in the author's opinion, in favour of the preparation of "polyvalent" sera. During last year at the British Institute of Preventive Medicine Farm at Sudbury, streptococci from two sources have been used—(1) from an acute abscess, and (2) from a fatal case of acute septicaemia. No. 1, after its virulence is exalted by the passage method through rabbits, is so powerful

that one-millionth of a cubic centimetre of liquid culture kills a rabbit in twenty-four hours.

If a bactericidal serum has to be used, one of even a more compound nature might with advantage be prepared.

Van de Velde suggested that as the agglutinating action of a serum on a particular race of streptococcus is in direct proportion to its bactericidal property, cultures of streptococci from various sources should be tested against the blood of the patient. These streptococci could have their corresponding sera prepared. The serum which corresponded to the organism which agglutinated in the shortest time, by the action of the patient's blood, would be the one most likely to stem the progress of the disease.

There is no doubt, however, that a "polyvalent" serum is superior to this method of treatment. In the course of a streptococcal infection, some time must elapse before the different bactericidal substances are formed in the blood which exert an agglutinating action on the particular race of streptococcus concerned, and the longer treatment is delayed the less likely is a favourable termination.

It has also been clearly shown that there is good reason for the belief that the strength of an antistreptococcus serum is extremely liable to diminish if it be kept for any length of time. In the sera first prepared, a little phenol was added to preserve them. This might account for the diminution in these cases. There is now no antiseptic added, and any impairment of the strength of a serum must be due to the effect of physical or chemical agencies on an unstable compound. An interesting fact in regard to this was shown by the different behaviour of Marmorek's serum in the hands of different observers. The good results from this serum were all obtained by workers in France. It seems probable that these were able to obtain fresher supplies of the serum than those beyond its borders. The impairment of the strength of the serum is also demonstrated in the results of observers who used two sera, one prepared by themselves and the other obtained from another source. In most instances positive results were obtained by their own sera, and

unsatisfactory or negative results by the other serum. Their own sera were probably prepared more recently than the other.

The author has endeavoured to prove that although the efficacy of the antistreptococcus serum in certain cases is very marked, yet its applicability is of a limited nature. The lines have also been suggested along which a serum might be prepared which would be more comprehensive in its action. The good results which follow its use are mainly seen in its effects on the local condition of the throat, in nasal discharges at an early stage of the illness, and on the glandular system. The invasion of the glandular system by an inflammatory process does not seem to be affected by the serum, as, in some cases, glandular enlargement took place after the serum was exhibited, and in other cases, where glandular infiltration was present, this became even more marked after the serum was used. There is considerable reason for believing, however, that the serum exhibits an inhibitory influence on the process of suppuration, as in no case of the series in which streptococci predominated did pus formation take place, although in many cases the inflammatory process was very acute. The change in the condition of the throat was often more marked, and showed itself more quickly than an improvement in the general condition. This would seem to indicate the necessity for an antitoxic rather than a bactericidal serum. The throat is the manufacturing place of the streptococcus toxine. This appears to be a very diffusible poison, and although the streptococci in the throat are presumably rendered harmless by the local action of the serum, the general circulation has received from the lymphatics of that region a sufficient dose of the toxine to lead to great depression. This toxine in the circulation is of course not affected by the serum. In many cases the extreme depression lasted till the throat exhibited healthy granulating ulcers. The effect of the serum on the temperature must be described as unreliable. It is impossible to attribute to the serum any influence on either the onset or the course of albuminuria occurring in this affection, as nephritis does not

seem to be commoner with a severe attack of scarlet fever than with a mild one.

The author would therefore submit that, as in the various complications of scarlet fever there may be a plurality of races of streptococci concerned, or a different race of streptococcus connected with each case, a "polyvalent" serum has much to recommend it.

There is no doubt that in the treatment of septicaemic conditions, the hope of the future lies in the discovery of a streptococcus antitoxin. The most potent antimicrobial serum yet prepared does not approximate to the power of the antidiphtheritic serum against the toxine of the bacillus diphtheriae. The efforts of laboratory workers must be in the direction of preparing a more powerful and stable serum.

The serum being then of a higher bactericidal value, the quantity necessary to be injected might be lessened. It has been found, by preparing antidiphtheritic serum of higher "immunization value" and thereby lessening the quantity injected, that the cutaneous disturbances incidental to its use have been less frequent. Similar good results might follow an increase in the potency of antistreptococcus serum.

BIBLIOGRAPHY.

- Klein.—*Micro-Organisms and Disease*, 1896.
 Sternberg.—*Text Book of Bacteriology*, 1897.
 Abbot.—*Principles of Bacteriology*, 1891.
 Hewlett.—*A Manual of Bacteriology*, 1898.
 Crookshanks.—*Bacteriology and Infective Diseases*, 1896.
 M'Farland.—*Text Book upon the Pathogenic Bacteria*, 1896.
 Muir and Ritchie.—*Manual of Bacteriology*, 1897.
Annales de l'Institut Pasteur, July, 1895, "Le Streptocoque et le Sérum Antistreptococcique," par le Dr. Alexandre Marmorek.
 H. Van de Velde.—"De la Necessité d'un Sérum Antistreptococcique Polyvalent pour combattre les Streptococcies chez le Lapin," *Archives de Médecine Expérimentelle*. Paris, 1897.
Archives des Sciences Biologiques, 1896, No. 5, p. 405.
Gazette hebdomadaire de Médecine et de Chirurgie, 1896, No. 64, p. 757.

- Roger.—“Nouvelles Recherches sur le Streptocoque,” *Gazette Médicale de Paris*, 1895 ; *Presse Médicale de Paris*, 1895.
- C. Parascandolo.—“Expériences Séro-thérapeutiques contre les Infections par les Microbes Pyogènes et contre l'Erysipèle, *Archives de Médecine Expérimentelle*, 1896.
- “Streptococcus Pyogenes and Antistreptococcus Serum.”—R. W. Bulloch, *Lancet*, May 2nd, 1896.
- B. Borneman.—“Ueber das antistreptokokken Serum,” *Wiener klinische Wochenschrift*, 1896.
- M. Méry.—“Sur une Variété de Streptocoque Réfractaire au Sérum Marmorek, *Comptes Rendus de la Société de Biologie*, 1896.

SERIES No. I. CASES IN WHICH THE THROAT SYMPTOMS WERE PROMINENT BUT WERE NOT
ATTENDED WITH EXCESSIVE DEPRESSION.

No.	Age in years or fraction of year.	Day of illness when first in- fection made.	Total quantity of β1 serum used.	BACTERIOLOGICAL EXAMINATION.	REMARKS.
I.	4	3rd	10 cc.	Streptococci very abundant. Staphylococci also present.	Rash somewhat patchy over arms and legs. Deep ulcers with unhealthy-looking edges over tonsils and pharyn- geal wall. No ulceration in front of the anterior pillars of the fauces. No nasal discharge. Slight bilateral cervical adenopathy.
II.	9	6th	10 cc.	Both streptococci and staphylo- cocci present, the former very abundant in nasal discharge.	Bright scarlet rash over trunk and extremities, somewhat patchy over buttocks. Ulceration over both tonsils and uvula. Seropurulent nasal discharge. Anterior segment of mouth clean and free from ulceration. Moderate bilateral cervical adenopathy.
III.	6	11th	20 cc.	Streptococci very abundant in throat. Staphylococci also present.	Throat very dirty with ulcers on dorsum of tongue and inside of cheeks and gums. Profuse seropurulent nasal discharge. Double otorrhoea. Moderate bilateral cervical adenopathy.

SERIES No. II. CASES IN WHICH THE THROAT SYMPTOMS WERE PROMINENT AND WERE
ATTENDED WITH GREAT DEPRESSION.

IV.	8	16th	60 cc.	Staphylococci, diplococci, and streptococci present in throat and mouth. Diplococci very abundant in ear discharge.	Rash patchy over buttocks and lower extremities. Throat extensively ulcerated, implicating tonsils, anterior pillars of the fauces, uvula, and pharyngeal wall. Pro- fuse seropurulent nasal discharge. Moderate bilateral cervical adenopathy. Right otorrhoea. Death on 30th day of illness.
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V.	5½	4th	30 cc.	One or two small chains of streptococci from gelatine culture. Practically a pure culture of staphylococcus pyogenes aureus.	Frank punctate scarlatinal rash over both trunk and extremities. Throat extensively ulcerated. Profuse nasal discharge. Ulceration proceeded very rapidly, as evidenced by marked huskiness and dysphagia. Slight cervical adenopathy. Septic rash on 8th day of illness over elbows, knees, and ankles. Death on 11th day of illness.
VI.	7	5th	20 cc.	Streptococci, staphylococci, and a short rod (probably bacillus coli communis) recovered from gelatine & agar cultures. Streptococci very abundant. Streptococci very abundant. Staphylococci also present.	Well marked scarlet punctate rash. Both tonsils extensively ulcerated. Slight discharge from right nostril only, lasting for one day. Slight bilateral cervical adenopathy. No otorrhoea.
VII.	2	3rd	50 cc.		Typical punctate scarlatiniform rash universally distributed. Large excavated ulcer present over left tonsil. Profuse seropurulent nasal discharge. Later the ulceration spread all over the throat and extended forward over the hard palate; also involved the gums and the inside of the cheeks. Bright septic rash on the 9th day of illness over elbows and knees. Severe cervical adenopathy. Temperature half an hour before death 105°·2. P.M. temperature 106°·8.
VIII.	3	4th	30 cc.	Streptococci and staphylococci both recovered from gelatine and agar cultures. Staphylococci very abundant. Streptococci not present to any extent.	Punctate scarlatiniform rash universally distributed. Both tonsils, anterior pillars of fauces, and uvula ulcerated; also soft tissues over hard palate and inside of cheeks and gums. No nasal discharge. Moderate bilateral cervical adenopathy. Marked rigidity of lower extremities some days before death. Death on 62nd day of illness.
IX.	6½	4th	10 cc.	Almost pure agar and gelatine culture of streptococci. Staphylococcus pyogenes aureus present, but few.	Considerable ulceration of the throat. Both anterior pillars of fauces completely eaten through, and through the opening thus formed on the left pillar a large deep excavated ulcer seen on the corresponding tonsil. The tongue studded with a number of small excavated ulcers. Moderate bilateral cervical adenopathy. Left otorrhoea.

No.	Age in years or fraction of year.	Day of illness when first injection made.	Total quantity of β 1 Serum used.	BACTERIOLOGICAL EXAMINATION.	REMARKS.
X.	3	18th	20 cc.	Staphylococci, diplococci, streptococci, and a short rod (probably bacillus coli communis) recovered from throat.	On admission diagnosis not confirmed. Nine days after admission a well marked punctate rash appeared over the trunk and extremities, with morning temperature 103.4°. Ulceration over both tonsils and uvula. Profuse nasal discharge. On 7th day after appearance of rash a well marked septic rash noticed over dorsal surfaces of hands and feet. Ante-mortem temperature 108.2°. Death on 26th day of illness.
XI.	4	10th	30 cc.	Abundance of streptococci recovered from throat. In the aural discharge bacillus tetragonus & diplococci also found.	Well marked rash over trunk and extremities. Both tonsils ulcerated, also anterior pillar of fauces and uvula. Discharge from both nostrils. Slight bilateral cervical adenopathy. Left otorrhoea.
XII.	5½	23rd	20 cc.	Streptococci and staphylococci present, the former abundant.	A very sharp case of scarlet fever. Rash patchy, almost morbilliform over thighs on outer and posterior aspects. Ulceration of soft palate and uvula began soon after admission, and extended rapidly. Perforation through the soft palate on the left side. Severe bilateral cervical adenopathy. Double purulent otorrhoea.
XIII.	4	4th	80 cc.	Streptococci and staphylococci present, the former very abundant.	Faint general punctate rash. Ulceration over both tonsils and anterior pillars of fauces. Nasal discharge sero-purulent in character. Very grave angina present with delirium and restlessness. Moderate cervical adenopathy.
XIV.	4	9th	65 cc.	Streptococci and staphylococci present, the former very abundant.	General punctate rash well marked. Severe bilateral adenopathy. Ulceration over both tonsils and back of pharynx. No nasal discharge.
XV.	8	13th	40 cc.	Streptococci and staphylococci present, the former predominating.	Rash general, punctate. Considerable ulceration over both tonsils. Slight cervical adenopathy. No nasal discharge. No otorrhoea.

XVI.	4	6th	60 cc.	Staphylococci and streptococci present, the former more numerous.	On admission rash fading on legs and chest. Extensive ulceration of both tonsils, uvula, and back wall of pharynx. Seropurulent discharge from nose. Severe diarrhoea all through illness. Moderate bilateral cervical adenopathy. Death on 18th day of illness.
XVII.	4	11th	20 cc.	Mixed infection with staphylococci and streptococci, the former predominating.	Case of very severe angina. Extreme ulceration of both tonsils and soft palate. Copious seropurulent nasal discharge. Persistent diarrhoea all through illness. Lungs involved latterly. Moderate bilateral cervical adenopathy. Death on 12th day of illness.
XVIII.	4	7th	160 cc.	An almost pure staphylococcus infection—a few streptococci present in the field.	Rash well developed, punctate, universally distributed. Extensive ulceration of both tonsils, especially right. Profuse discharge from nose, seropurulent. General enlargement of lymphatic glands in groins, axillae, and triangles of neck. Acute nephritis. Severe diarrhoea, which persisted to the end. Death on 34th day of illness.
XIX.	3½	6th	40 cc.	Streptococci and staphylococci present, the former outgrowing the latter.	Rash well out on the face, simulating measles; on the body typically punctate in character. Extensive ulceration of both tonsils and soft palate. Nasal discharge abundant, seropurulent. Moderate bilateral cervical adenopathy.
XX.	3	6th	20 cc.	Streptococci and staphylococci both recovered from throat.	Rash has faded, leaving injected red points on front of legs and on extensor surfaces of the arms. Profuse purulent nasal discharge. Diarrhoea severe and persistent. Ulceration over entire throat leading to perforation of the left velum palati. Double otorrhoea. Moderate cervical adenopathy on both sides.
XXI.	2	9th	30 cc.	Streptococci and staphylococci present, the former predominating.	A fading general scarlet rash, papular over external aspects of legs. Severe angina present. Profuse purulent nasal discharge. Ulceration over opposing surfaces of tonsils; also on gums and inside of cheeks. Moderate cervical adenopathy.

No.	Age in years or fraction of year.	Day of illness when first in- fection made.	Total quantity of β1 Serum used.	BACTERIOLOGICAL EXAMINATION.	REMARKS.
XXII.	3½	11th	30 cc.	Streptococci and staphylococci recovered from throat.	Rash typical and generally distributed. Throat structures extremely dirty-looking, with deep ulcers on opposing surfaces of tonsils, and over soft palate. Profuse sero- purulent nasal discharge. Inside of cheeks and gums in- volved in ulcerative process. Suppurative arthritis of right elbow joint. Double otorrhoea. Moderate cer- vical adenopathy.
XXIII.	6	7th	40 cc.	Streptococci and staphylococci both recovered from throat.	Extensive ulceration on right tonsil and on uvula. Pro- fuse seropurulent nasal discharge. Moderate cervical enlargement. Delirium present. Left otorrhoea.
XXIV.	6	8th	70 cc.	Streptococci and staphylococci both recovered from throat, the former predominating.	Typical scarlet rash on trunk and extremities. Sloughy areas over both tonsils and soft palate. Seropurulent nasal discharge. Moderate bilateral cervical adeno- pathy. Left otorrhoea.
XXV.	3	2nd	30 cc.	Staphylococci and streptococci, the former very abundant.	Rash well out and characteristic. Fauces, palate, and tonsils much injected, the latter being ulcerated. Copious seropurulent nasal discharge. Moderate en- largement of submaxillary glands. Death 3 days after admission, on 5th day of illness.
XXVI.	2½	10th	20 cc.	Streptococci and staphylococci present, both abundant.	Rash "went in" very quickly. Extensive faucitis and ulceration of both tonsils, uvula, and soft palate. Foul smelling nasal discharge. Severe angina present. Slight cervical adenopathy. Death on 11th day of illness.
XXVII.	3	10th	80 cc.	Streptococci and staphylococci present, the former much more numerous.	Typical punctate rash. Severe ulceration over both tonsils and uvula. Gums, inside of cheeks, and lips also involved in ulcerative process. Serous nasal discharge. Slight cervical adenopathy. Violent delirium present. Double otorrhoea. Inflammation of left eye, lachry- mal abscess formed.

No rash present but well marked desquamation. Extreme ulceration over both tonsils, uvula, anterior pillars of the fauces, and posterior wall of pharynx; also implicates gums and inside of cheeks. Severe bilateral cervical adenopathy. No nasal discharge. Death on 16th day of illness.

Patchy punctate rash universally distributed. Tendency to assume papular form in parts. Great enlargement of cervical glands with brawny infiltration of the cellular tissue of the neck. No nasal discharge. No otorrhoea. Death on 12th day of illness.

No rash made out. Ulceration on left side of soft palate. Profuse seropurulent nasal discharge. Diarrhoea persistent. Moderate bilateral cervical adenopathy. No otorrhoea.

Mixed infection. Streptococci few in number. Staphylococci very plentiful.

60 cc.

12th

3

XXVIII.

Mixed infection. Staphylococci much more numerous than streptococci.

70 cc.

7th

5

XXIX.

Streptococci and staphylococci present, the former very plentiful.

20 cc.

4th

1⁵/₁₂

XXX.

SERIES No. III. CASES CHARACTERIZED BY EARLY ULCERATION OF THE THROAT, AND ATTENDED WITH LIVID AND PATCHY RASHES. EXTREME DEPRESSION A MARKED FEATURE.

Rash universally distributed; somewhat papular over back; markedly livid in colour. Extensive and deep ulceration of right tonsil. Moderate cervical adenopathy. Double otorrhoea very profuse. No nasal discharge.

Punctate livid rash universally distributed. Extensive and deep ulceration over both tonsils and anterior pillars of fauces. Slight nasal discharge. Double otorrhoea. Severe cervical adenopathy.

Livid and patchy rash. Ulceration, very extensive, of both tonsils and uvula. Profuse seropurulent nasal discharge. Extreme diarrhoea all through illness. Bronchopneumonia supervened before death. Moderate cervical adenopathy. Death on 17th day of illness. Ante-mortem temperature 107.4°.

Streptococci and staphylococci present, the former very abundant.

40 cc.

12th

3

XXXI.

Streptococci and staphylococci both recovered from throat, and from gelatine & agar tubes. Streptococci very abundant.

70 cc.

9th

6

XXXII.

Staphylococci, diplococci, and streptococci recovered. Staphylococci outnumber other organisms.

100 cc.

6th

2

XXXIII.

No.	Age in years or fraction of year.	Day of illness when first infection made.	Total quantity of β 1 Serum used.	BACTERIOLOGICAL EXAMINATION.	REMARKS.
XXXIV.	2	6th	20 cc.	Practically a pure infection of staphylococcus pyogenes aureus. One chain is found in two or three cover-glass preparations.	Livid and patchy rash over extremities; on trunk, bright scarlet and confluent. Ulceration over right tonsil and right anterior pillar of the fauces. Profuse nasal discharge. Very severe bilateral cervical adenopathy. Death on 9th day of illness.
XXXV.	6	10th	20 cc.	Streptococci and staphylococci present, the former very abundant.	Scarlatina anginosa of a very asthenic type. Rash patchy and livid over lower extremities. Deep ulceration over both tonsils. Profuse purulent nasal discharge. Moderate bilateral cervical adenopathy.
XXXVI.	4	9th	30 cc.	An almost pure infection of streptococci.	Rash punctate, patchy, and livid over thighs. Extreme ulceration over both tonsils and soft palate. Copious seropurulent discharge from nose. Moderate enlargement of the submaxillary glands. Death on 28th day of illness.
XXXVII.	2½	8th	60 cc.	An almost pure infection of streptococci, associated with diplococci.	Rash livid over lower extremities; opposed surfaces of tonsils deeply ulcerated. No nasal discharge. Slight enlargement of submaxillary glands. Extensive bronchopneumonia in right lung before death. Death on 28th day of illness.
XXXVIII.	4	4th	20 cc.	Staphylococci and streptococci recovered from throat.	Livid rash which ultimately became petechial. Profuse mucopurulent discharge from nose. Severe diarrhoea from the onset. Severe enlargement of submaxillary glands. Extreme angina. Death on 12th day of illness.
XXXIX.	6½	5th	110 cc.	Mixed infection. Staphylococci, streptococci, and diplococci recovered from throat.	Rash universally distributed, blotchy and livid over forearms. Hectic flush over cheeks. Large necrotic patches over tonsils and soft palate. Profuse serous nasal discharge. Severe bilateral cervical adenopathy. Double otorrhoea. Severe angina present. Severe and persistent diarrhoea. Death on 28th day of illness.

XL.	$3\frac{1}{2}$	6th	20 cc.	A pure staphylococcus infection. No streptococci nor Loeffler's bacilli present.	Rash over face is somewhat morbilliform. Severe cervical adenopathy with infiltration of cellular tissue round about. Considerable purulent nasal discharge. Bronchopneumonia before death. Death on 10th day of illness.
XLI.	7	11th	20 cc.	Mixed infection. Staphylococci, diplococci, and streptococci recovered from cultures.	No rash observed, but well marked desquamation over hands and feet. Deep and extensive ulceration of throat structures. Profuse purulent nasal discharge. Septic rash on extensor surfaces of elbows, knees, and over front of both ankles on 12th day of illness. Moderate enlargement of submaxillary glands. Death on 20th day of illness.
XLII.	$1\frac{3}{4}$	8th	80 cc.	Mixed infection. Staphylococci outnumbering streptococci.	Typical rash present. Considerable faucitis, with ulceration over left tonsil. Moderate enlargement of submaxillary glands. Serous nasal discharge. Extreme angina present. Death on 29th day of illness.

SERIES No. IV. CASES IN WHICH THERE WAS NO MANIFEST ULCERATION OF THE THROAT, BUT WHICH WERE ATTENDED WITH A PROFUSE NASAL DISCHARGE.

XLIII.	$1\frac{1}{2}$	6th	10 cc.	Streptococci and staphylococcus pyogenes aureus both present. Practically a pure culture of streptococci recovered from nasal discharge.	Sparse eruption somewhat livid and patchy over the trunk. Throat covered with exudation. Profuse seropurulent nasal discharge. Double purulent otorrhoea. Diarrhoea to the extent of five motions per diem. Moderate enlargement of submaxillary glands.
XLIV.	$1\frac{2}{3}$	13th	20 cc.	Staphylococci, streptococci, and diplococci, recovered from throat. Streptococci very abundant.	Rash typical and generally distributed. Mouth and throat covered with a dirty exudation. Profuse seropurulent nasal discharge. Diarrhoea to the extent of four motions per diem. Moderate enlargement of submaxillary glands.
XLV.	5	16th	30 cc.	Streptococci and staphylococci, the former very abundant.	Bright scarlet rash. Profuse seropurulent nasal discharge. Enlargement of glands at angles of jaw. Throat covered with dirty exudation. No ulceration observed.

No.	Age in years or fraction of year.	Day of illness when first injection made.	Total quantity of β 1 Serum used.	BACTERIOLOGICAL EXAMINATION.	REMARKS.
XLVI.	5½	3rd	30 cc.	Streptococci abundant. Staphylococci also present.	Rash bright, patchy, and raised on the limbs, simulating measles; on the trunk, more confluent. Respiration characteristic of nasopharyngeal obstruction. Well marked seropurulent nasal discharge. Moderate enlargement of glands at angles of jaw. Throat dirty, but no evidence of ulceration. Double otorrhoea.
XLVII.	5	20th	150 cc.	Streptococci very numerous in throat. Cover-glass preparations were made directly, and agar and gelatine tubes inoculated with the pus from cervical abscesses. Both streptococci and staphylococci were recovered, the former being very abundant.	<p>The following case was admitted suffering from a mild attack of Scarlet Fever. A measles rash appeared on the eighth day of residence in hospital. The case thereafter assumed a severe character.</p> <p>Patient developed measles on 8th day of residence in hospital. Bronchopneumonia supervened two days after appearance of rash. Extreme ulceration of right anterior pillar of the fauces, both tonsils and uvula. Severe cervical adenopathy, which went on to suppuration. No nasal discharge. Death from general tuberculosis.</p>

The author acknowledges his indebtedness to Drs. Brownlie, M'Harg, M'Coll, Mackay, and Frew—his colleagues on the staff of the Hospital—for careful notes taken of several of the cases in the above series.

ON THE ADMINISTRATION OF NITROUS OXIDE AND OXYGEN FOR INDUCING ANAESTHESIA.

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THE administration of nitrous oxide, combined with regulated percentages of oxygen, has within recent date been receiving more general recognition as an anaesthetic of much value.

It has the advantage over nitrous oxide alone of producing a more tranquil anaesthesia, lasting some fifteen or twenty seconds longer, and unattended with the phenomena of asphyxia. In addition, when the operation is not in the neighbourhood of the mouth, we can continue to give the combined gases till the operation is completed; while, when nitrous oxide alone is employed, the inhalation must be stopped as soon as signs of asphyxia occur.

I propose to describe the apparatus employed and its method of use, and, lastly, to consider the various forms of operation in which it may be employed.

The apparatus used for the administration of nitrous oxide and oxygen is that devised by Dr. Frederick W. Hewitt, of London, and made for him by Messrs. George Barth & Co., Oxford Street.

In describing the apparatus I shall draw largely from Dr. Hewitt's little book on the administration of nitrous oxide and oxygen.

Figure 1 represents the complete apparatus, consisting of two nitrous oxide cylinders, one oxygen cylinder, a combined

stand and union, double india-rubber tubes (one running inside the other) for conducting the two gases from the cylinders to the bags, two india-rubber bags joined together by a septum common to both, a combined regulating stop-

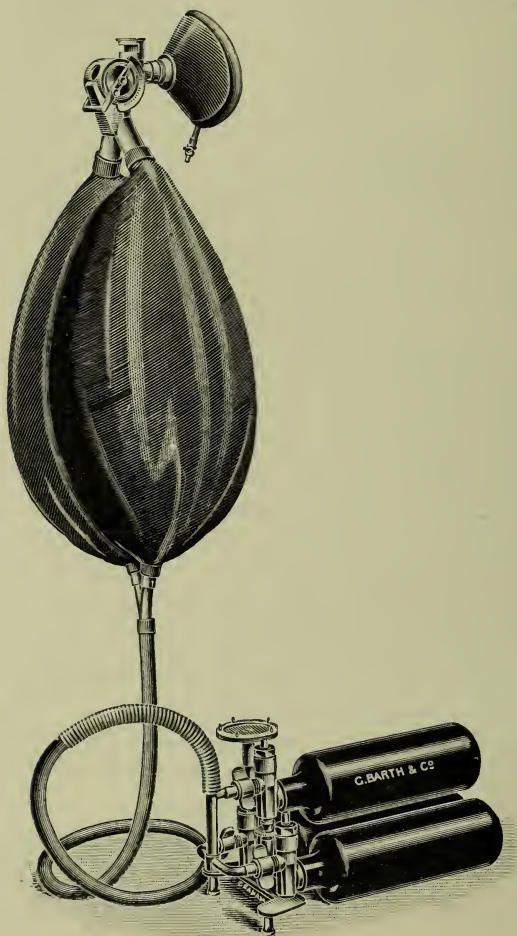


FIG. 1.

cock and mixing chamber, and a face piece. Each nitrous oxide cylinder will furnish 50 gallons of nitrous oxide gas; and each oxygen cylinder about 15 gallons of oxygen. When the foot key is placed upon one of the nitrous oxide cylinders,

and is turned, the liberated nitrous oxide passes to its bag through brass and india-rubber tubes of comparatively large calibre. When oxygen is similarly released from its cylinder, it passes to its bag through brass and india-rubber tubes, which are so much smaller than the nitrous oxide tubes that they are made to travel inside the latter.

The regulating stop-cock and mixing chamber are shown in detail in Figure 2.

The nitrous oxide bag is attached to the tube NOT, the orifice of which NOO is shown. The oxygen bag is attached to the tube OT, which communicates above with a little oxygen chamber OC. There are ten minute holes between the oxygen chamber OC and the mixing chamber. Only three of these ten holes OO are shown in the figure. The tubes OT and NOT are furnished with removable valves, *iv* and *iv'*, which act during inspiration, and which prevent diffusion between the gases of the two bags. AH is the air hole; IV is the main inspiratory valve; EV is the expiratory valve with its chimney C; PD shown in

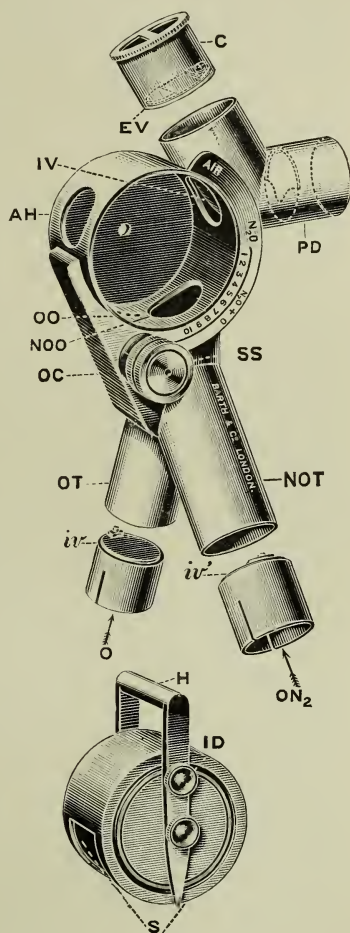


FIG. 2.

dotted outline is a partial diaphragm mounted upon a removable inner tube which serves to direct the expirations towards the expiratory valve EV. To the circumference of the stop-cock and mixing chamber is fixed a flange with AIR, N_2O , and $N_2O + O$ engraved upon it.

There are also figures from 1 to 10 inclusive belonging to the N_2O+O part of the flange. When the indicator of the handle H points to AIR, as in Fig. 1, the slot s of the drum ID allows air to pass through AH and IV during the act of inspiration; but by reason of the other part of the drum covering the orifices NOO and OO, nothing but air is breathed. When the indicator is moved to N_2O , the drum closes AH and opens NOO, the oxygen orifices still remaining covered. Pure nitrous oxide is therefore inhaled. When the indicator reaches I on the N_2O+O part of the flange, the nitrous oxide orifice NOO still remains open, but in addition one of the oxygen orifices OO becomes uncovered by the revolution of the inner drum. When 2 is reached two oxygen holes are open, and so on up to 10, the nitrous oxide orifice remaining patent throughout.

Having described the apparatus, I shall briefly speak of the administration of the combined gases. Patients require no special preparation, as in the giving of chloroform, but it lessens the chances of sickness (which is extremely rare) if no food be taken for three or four hours previous to the taking of the gas. Of course, if a long anaesthesia is required this is all the more essential. The bladder should be empty. All clothing should be loose. Stays, bands, collars, etc., ought to be undone. In the case of any operation on the mouth or throat it is necessary to use a gag, which is inserted before the patient is anaesthetized. The bags (oxygen and nitrous oxide) having been half filled with their respective gases the face piece is applied, the patient being in the sitting posture, with his head resting on the back of the chair, or if a couch is used the head ought to be raised by several pillows. It is well to have several face pieces, for accuracy of application to the face is essential. Two masks, one medium size and the other the smallest procurable, are generally sufficient. The patient, unless a child, should be told to breathe freely as soon as the face piece with the indicator pointing to AIR is put on. The valves should be heard acting, as otherwise the face piece is not fitting accurately. Having satisfied yourself that the breathing is regular, the indicator is turned to 2. The patient is

now breathing nitrous oxide with two per cent. of oxygen. The foot key of the nitrous oxide cylinder should be turned on slightly so as to replace the nitrous oxide which is rapidly inhaled by the patient. It is of the utmost importance to keep the nitrous oxide and the oxygen bags constantly at the same size, viz., half filled. At first we increase the oxygen very slowly, generally allowing the patient to take two or three breaths between each number. In young children and feeble adults the amount of oxygen can be quickly increased till, after the first forty seconds, 7 to 10 per cent. of oxygen is given. A strong, healthy adult rarely requires more than 5 or 6 per cent. of oxygen. We have to guard against the giving of too much or too little oxygen; as Dr. Hewitt puts it, the anaesthetist has to avoid the Scylla of asphyxia on the one hand and the Charybdis of excitement on the other, that is, if too much oxygen be given we get excitement mental and muscular (laughter, shouting, kicking, and struggling), whereas if too little oxygen be given, then lividity, stertor, and muscular twitchings result. The absence of any indications of excitement on the one hand and asphyxia on the other will prove that the proportions of the two gases are properly adjusted. The average period of inhalation required to produce anaesthesia varies; Dr. Hewitt gives it at 110.5 seconds. From a very limited experience, I should feel inclined to put it at rather less. The signs of anaesthesia being fully established are the *loss of conjunctival reflex, breathing regular or slightly snoring in character, and flaccidity of the extremities*. In some cases the conjunctival reflex is not completely lost, but there is always a relaxed condition of the eyelids and a fixed condition of the globes, which indicate the approach of the anaesthetic state. If the operation requires the removal of the face piece, as in teeth extraction, we naturally wish to have anaesthesia fully established before doing so, and the more especially if an attempt is to be made to remove several teeth. Hewitt states that the average duration of the anaesthetic stage is about 44 seconds, but in many cases a much longer period may be obtained. I have frequently obtained a period allowing of the removal of from four to six

teeth, provided no difficulty was experienced in their extraction. Experienced dentists tell me they are quite satisfied that it increases the available period of anaesthesia as compared with the nitrous oxide alone.

For the sake of convenience the uses to which nitrous oxide and oxygen may be put will be considered under the following three groups:

1. Its employment for short operations.
2. For long or comparatively long operations.
3. Its use previous to the giving of ether.

1. *In Short Operations.*—Nitrous oxide and oxygen is *par excellence* the best anaesthetic that can be employed, for anaesthesia is rapidly induced, the after effects pass away quickly, and there is practically no risk to life.

The operation in which it is most constantly employed is of course teeth extraction, and as the mask has to be withdrawn as soon as anaesthesia is established the period of anaesthesia is necessarily short, rarely lasting more than some forty or fifty seconds. In another operation about the mouth, the removal of tonsils and adenoids, it is a most convenient anaesthetic, but, of course, implies rapid operation. I have for some months back given the combined gases to patients of Dr. John Macintyre. They have all been comparatively young children, and the results have been entirely satisfactory. The patient, seated in a chair, is put under the influence of the gas, the mouth opened quickly with a Mason's gag, and both tonsils and adenoids removed. As soon as the operation is completed the head is depressed forwards so as to allow of the blood flowing from the mouth and nostrils. It has the immense advantage over chloroform in that anaesthesia is rapidly induced, and as the patient comes out of the anaesthetic quickly there is no complication from blood trickling into the air passage and causing asphyxia. There is rarely, too, any nausea or after sickness. As I have already said the period of anaesthesia available for operation is a short one, but even if the little patient feels the latter part of the operation there is no special disadvantage. After all the freedom from any risk to life is its chief claim for employment.

For many other minor surgical procedures the combined gases are eminently suitable. One could give a list of cases in which it may be employed, but that is unnecessary. Among the more common may be mentioned bending of stiff joints, opening superficial and deep abscesses, passing catheters, applying the actual cautery, forcibly dilating the anterior nasal passages, and setting fractures. Its advantage in cases where the operation is not about the mouth is apparent, for the inhalation of the gases is gone on with till the operation is completed—some operations requiring an anaesthesia of say five minutes or so can in this way be readily performed. It is undoubtedly difficult to regulate the percentage of oxygen, but in the event of any undue lividity from insufficient oxygen the removal of the face piece now and again causes the lividity to disappear. The difficulty which can only be overcome by experience is the giving too much or too little oxygen, for if the former results then we have excitement manifested by screaming and struggling, whereas if too little oxygen be given then lividity and clonic spasms may ensue. Still, in operations limited in their duration these difficulties are comparatively rarely encountered, and a little practice would prevent their occurrence.

2. *For Long or comparatively Long Operations.*—Mr. Bellamy Gardner, of London, was the first to employ gas and oxygen for prolonged periods, and in the *Lancet* for June 12, 1897, published cases in which successful anaesthesia was maintained for over fifteen minutes, thus allowing of major operations being performed. Since then, Mr. H. Patterson, of St. Bartholomew's Hospital, has described cases in which anaesthesia was kept up for nearly an hour, permitting amputations of limbs and excisions of joints to be performed. The longest period I have ever given it for was thirty-five minutes in a case of tumour of the breast excised by Dr. Rutherford. Anaesthesia was readily effected and maintained without difficulty, and within a comparatively short time after the withdrawal of the face piece consciousness returned. No sickness followed. I cannot, however, believe that nitrous oxide and oxygen will ever be generally employed for long operations. Surgeons as

a rule in major operations desire profound anaesthesia, and that is what one cannot guarantee for any length of time. It is true that its chief claim for employment—its absolute safety as regards risk to life—might induce anaesthetists to consider its claims, but the difficulty of maintaining efficient anaesthesia and the consumption of large quantities of nitrous oxide (100 gallons will last about half an hour) will militate against its employment unless under special circumstances.

3. *Lastly, Nitrous Oxide and Oxygen may be given previous to the Employment of Ether.*—Many of the London anaesthetists advocate its use, as it allows the patient to be anaesthetized without any of the disagreeable initial effects of ether. In nervous subjects especially it is of great advantage, for you get rapid loss of consciousness, and as the patient, as soon as he is under the influence of the gas, breathes regularly and deeply, we have none of the holding of the breath and struggling so apt to occur in the initial stage of etherization.

The gas and oxygen are given until anaesthesia occurs, and then an Ormsby inhaler with its sponge charged with ether is substituted, and anaesthesia gone on with in the usual way. Nitrous oxide alone is usually recommended in preference to the combined gases, but I find that with the addition of oxygen you can get a more thorough degree of anaesthesia before applying the ether mask.

In conclusion I would urge the claims of nitrous oxide and oxygen for at anyrate minor surgical procedures. Its practical freedom from any risk to life, the rapidity of anaesthesia, and the absence of after effects must surely appeal to surgeons. Perhaps its disadvantages, namely, initial cost of apparatus, trouble of getting the gas bottles refilled, and unacquaintance with the methods of administration, will prevent its general adoption.

THREE EXAMPLES OF PULMONARY EMBOLISM IN SURGICAL CASES.

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PULMONARY embolism in surgical cases is rightly regarded as being a somewhat rare condition, and I can call to mind only two instances where this was ascertained to be the cause of death in cases under my care during the many years I have held a hospital appointment, previous to those herein described. Yet the three cases now placed on record all occurred within two months, and two of them occupied the wards at the same time. I am, of course, excluding from consideration infarctions of the lung due to septic infection and to the dissemination of cancer, which are not by any means infrequent; and am confining my statement to embolism due to clots carried from peripheral veins, or to vegetations dislodged from diseased heart valves. The cases under discussion have only one feature in common, namely, that there were no clots found in the right heart or in the veins entering it. In other respects they show the widest variation, in symptoms, mode of onset, in the condition immediately leading up to the fatal issue, and in the lesions found at the post-mortem examination. I give the report of the cases, following each chronicle by a brief commentary, pointing out the interesting features.

CASE 1.

J. A., aged 32, carter, was admitted into Ward 25 of the Royal Infirmary on June 1st, 1899, complaining of pain

over the back part of the left lower ribs, the result of a blow received from the shaft of a cart. There was no loss of consciousness at the time of the accident, and no injury whatsoever of the head or spinal column. On examination at the seat of pain no fracture of the ribs or ecchymosis could be detected, but there was general tenderness due to bruising. He looked pale and ill, and his pulse was rapid and feeble, and for this reason he was kept in hospital. On the afternoon of the following day, when his wife came to see him he could not speak to her; he attempted to say something, but could not make himself understood. The attention of the House Surgeon was drawn to his condition, and it was then noticed that the right arm and right side of the face were paralysed, but he could move his right leg. At 7 p.m. of the same day the paralysis was found to have extended to the right leg. On the morning of the third day a fuller record of his case was taken. The paralysis of the face was found to be limited to the lower part, and did not implicate the orbicularis palpebrarum, corrugator, or occipito-frontalis, patient being able to close his eye and wrinkle his brow. The tongue when protruded pointed to the right side. Tactile sensation was normal in the paralysed limbs, thermal sensation was very defective in the right arm, patient being unable to distinguish heat from cold. Plantar reflexes were normal. Patellar reflex not elicited in either leg. When tested by the dynamometer the right hand raised the indicator to 65, and the left to 140. Patient's condition gradually improved from June 4th to July 19th. On the 12th July it was recorded that patient was able to move his right leg quite freely, and that he could speak so as to be understood. By the end of June he was able to walk about the Infirmary grounds, carrying his right arm in a sling. The right foot dragged slightly, but showed a satisfactory improvement from day to day. The ward journal, under date July 15th, states: "To-day is the first time that patient has been able to use right forearm and hand since the onset of the hemiplegic attack."

On July 16th he complained of pain over the lower part of

the abdomen, and this lasted for the next three days, being only slightly relieved by external applications, and the internal administration of castor oil and laudanum. On the 19th he was much worse, his respiration was hurried and somewhat shallow, his pulse rapid and feeble. Percussion over the lungs gave no positive dulness in any part, but it was noted that over the right lung at the base posteriorly the respiration was tubular, and there were cooing râles. There was a short cough without expectoration, but he did not complain of pain in the chest. There was no abdominal distension. His respiration on several occasions had much of the Cheyne-Stokes character. The temperature kept a little above normal, but its highest point was only 99.2° F.; pulse 150, feeble; respirations 52. The record of the 24th July adds difficulty of swallowing to the other symptoms, and on the 30th his tongue was coated with a brownish crust; there were evidences of rapid emaciation, and he passed urine and faeces involuntarily. Dyspnoea was no longer present, but he gradually sank, and died early on the morning of 1st August, exactly two months after admission.

The *post-mortem examination* was made by Dr. J. W. Findlay, and his report runs as follows:

Thorax.—Heart weighs $13\frac{1}{2}$ oz., and is much larger than normal. Aortic and pulmonary valves are competent. Mitral and tricuspid orifices are dilated, the former admitting four fingers and the latter six. Right auricle somewhat dilated. Wall of both ventricles thin and cavities dilated. Heart muscle pale and friable. In the left ventricle at the apex is a round, nodular, and almost pedunculated ante-mortem clot, while extending from and around this, and reaching to within an inch of the aortic valve, is from 2 to 3 square inches of white thrombus. This is intimately associated with the heart wall, and cannot be separated without lacerating the cardiac muscle. This clot measures in thickness from $\frac{1}{4}$ to $\frac{3}{4}$ inch. Towards the left of the clot and beyond it there are numerous red thrombi within the muscular wall of the heart. Immediately above the aortic curtains the aorta shows three cushion-like elevations, irregularly circular, about $\frac{1}{4}$ inch in

diameter, and placed directly above the centre of each curtain. The aorta for the first two inches shows some irregular thickening, but there are no white or yellow patches. Coronary arteries at their commencement and for a short distance show early atheromatous change in the form of yellow patches and thickenings. On microscopic examination of the heart a considerable degree of fibrosis is found to exist, patches of dense fibrous tissue appearing here and there between patches of heart muscle. The connective tissue cells as a whole are much increased in number. In the new-formed fibrous tissue there are very numerous haematoidin crystals, both between the fibres and in the protoplasm of the cells, and numerous newly formed blood vessels are found in the fibroid areas. No thrombi are found in the vessels of the heart, such clots as exist in the heart muscle being placed between the muscular fibres. The clot is a laminated mixed thrombus in which fibrin filaments, red corpuscles (not much altered in shape), haematoidin crystals, and leucocytes are found.

Lungs.—*Left* is normal. *Right* is adherent by means of slight recent fibrinous exudation. About one ounce of fluid blood is present in the pleural cavity. Three distinct haemorrhagic infarctions are seen in the lung. The lower half of the upper lobe is solid, and cuts like liver. A small white thrombus about the size of a pea is found in the branch of the pulmonary artery leading to that lobe, and behind this a more recent red thrombus. The whole of the middle lobe is consolidated, and a large clot about $\frac{3}{4}$ inch in thickness and $\frac{5}{8}$ inch in length is found occupying the branch of the pulmonary artery entering it. In the lower lobe a wedge-shaped area of infarction exists with two free surfaces. The area is $2\frac{1}{2}$ inches in depth, and its surfaces are, roughly, two inches square. A small white thrombus is found just proximal to the infarcted area.

Abdomen.—Mesenteric glands generally enlarged; most of them are hard and firm, and some have undergone calcareous degeneration. *Intestines* normal; no indications of former ulceration. *Stomach* a little dilated; mucous membrane

thickened. *Pancreas* cirrhotic. *Liver* shows marked fatty change. *Spleen* small and firm. *Kidneys* slightly granular.

Brain.—At the upper end of the Rolandic area of the left side is a distinct depression of the cortex, and the brain generally on the left side looks smaller than on the right. None of the arteries forming the circle of Willis show atheromatous change, and the medullary branches entering the perforated spaces seem perfectly normal. No embolism or thrombosis of any of these arteries can be detected, and the island of Reil as seen from the surface seems normal. On section the left cortex is found to be less vascular than the right, and the whole of the left corpus striatum and optic thalamus is found to be in a condition of yellow softening, and this extends downwards and forwards into the third frontal convolution.

Commentary.—The great feature of interest in this case is the fact that such dire results followed from such a slight injury. The blow on the side was so small a matter that it caused neither fracture of the ribs nor ecchymosis of the skin. There was no local swelling caused by it, and not much tenderness on palpation. Yet it was followed by serious haemorrhage into the left corpus striatum and optic thalamus with consequent right hemiplegia. We should not have been surprised at this result had the patient been double his age and his arteries in an advanced stage of atheroma. But he was only 32 years of age, and there was no evidence of disease in the arteries of the brain at the post-mortem examination. We must, however, note that he was a man who admittedly drank to excess, and it is probable that he had suffered from syphilis, although his history was not very clear on that point. Further, the aorta showed some atheromatous indications at its commencement, and so did the coronary arteries, and from these indications we are inclined to conclude that the cerebral arteries were not really healthy. It was not, however, as the direct result of the injury that the hemiplegia followed; an interval of about thirty hours elapsed, during which he was lying at rest in bed. It is probable that the haemorrhage commenced at the time of the injury, and it was only when it

reached some magnitude that it produced serious effects on the brain. The recovery from the hemiplegia was not unsatisfactory, and in the leg was distinctly good. A month after the accident he was able to walk about the Infirmary grounds with only a slight drag of the foot; his speech, though thick and obviously impeded, was intelligible, and he had no difficulty in finding the right words. The recovery of the arm was less satisfactory, and he was obliged to carry it in a sling when walking about. The flexion of the fingers and wrist was good in range, but feeble in quality, that of the elbow was very defective, while the movements of the shoulder were almost in abeyance. From the amount of recovery which took place we were not prepared to find such an extensive softening in the corpus striatum and optic thalamus.

At what date the formation of the thrombus in the left ventricle took place is not easily ascertained. The clot was very firmly adherent to the ventricular wall, and involved the meshes of the columnae carnaeae; further, more recent thrombus was found in the muscular wall, which was continuous with the white clot in the cavity of the ventricle. The patient's symptoms at the onset of his fatal illness were not referable to the heart or lungs, but were entirely abdominal. On the 19th July, however, the rapid breathing and the quick, feeble pulse were the prominent symptoms; there was a short cough without expectoration, but no complaint of pain in the chest. We may assume that at the date mentioned obstruction to the normal action of the heart and lungs had commenced.

As to the mechanism of the production of the pulmonary embolism, it is not easy to arrive at a satisfactory explanation. (1) We can at once put out of view the suggestion that clots were carried from the left heart throughout the whole systemic circle to reach the right lung—for in the process of being so carried it would inevitably have happened that some clots would have reached other organs such as the kidneys and spleen. Now the post-mortem report shows that there were no infarctions in any organs but the right lung. (2) There is just a possibility that a clot from the left side of the

heart might reach the right by the coronary arteries and veins, and the possibility of this is increased by the fact that there were haemorrhages into the wall of the left ventricle. (3) It has long been admitted that clotting may commence in the smaller pulmonary vessels without embolism, and that this may extend to the larger branches of the artery, even as far as the main pulmonary arteries passing to the lungs. This condition—spoken of by Wilson Fox as “Spontaneous pulmonary thrombosis”—has been found to take place in pneumonia, in rare cases of pleurisy, in a few cases of diabetes, and where there is atheroma of the smaller pulmonary vessels. Such cases are distinguished clinically by the fact that the fatal issue is not sudden but protracted, as it was in this case. There is reason to assume that the formation of clot in the left heart would lead to retardation in the circulation through the lungs, and if at the same time there was present some pleurisy (as there was), and possibly some superficial pneumonia, we can understand how readily clotting in the vessels would take place. Even decoloration of the clot, its adhesion to the vessel wall, and lamination are possible when so long a period as a fortnight elapses between the onset of the condition and the fatal issue. This theory fits in best with the gradual (instead of sudden) onset of the symptoms, and the absence of acute pain in the lung such as usually accompanies pulmonary embolism. At the same time it must be acknowledged that several of the clots—and especially that plugging the pulmonary artery of the upper lobe—looked very like true emboli.

The involvement of the right lung alone is a common feature of pulmonary arterial blocking, but it is unusual to get the upper lobe implicated. Virchow found in his experiments that he “never got the upper lobe affected except when mercury or other finely divided substances were injected.”

The medico-legal aspect of the case deserves a little consideration. As far as I am aware no question of responsibility in regard to the injury has been raised, and no demand for compensation in respect to the man's injuries and death has been made. It is quite conceivable that these may yet have

to be considered, and how shall we answer them? The law, I understand, holds that the person causing the injury is answerable for all the consequences of that injury, however remote they may be. It might be "law," but it certainly would not be "equity," to hold the driver of the cart, the shaft of which struck my patient, responsible for his death. Nor would it be just to make the employer of that driver responsible for pecuniary compensation to the relatives of the deceased. Whatever may be the strict legal condition, there must always be in this and similar cases a consideration of the trivial nature of the injury, and also of the "vulnerability" of the man injured.

CASE 2.

Mrs. R., aged 40. Admitted into Ward 26 of the Royal Infirmary on July 25th, 1899. Three days before admission the patient's left foot, without known cause, became cold and swollen, and she was unable to put her weight upon it. The following day the toes became of a bluish tint and the foot more swollen, and on the third day the whole of the toes and the distal two-thirds of the foot became of a purple colour. She stated that about a fortnight earlier she had tingling and numbness in the *right* leg, and that it became cold and she was unable to use it; the condition, however, soon passed off. While engaged in washing clothes, about ten days before admission, the tips of the fingers of both hands became blanched, they also tingled, and she had the sensation of "pins and needles"; this necessitated her stopping washing for the day, but she was all right next morning. In other respects she had enjoyed good health.

Condition on Admission (from the ward journal).—"The toes and distal two-thirds of the dorsum of the foot are of a purple colour, the condition being more marked in the toes than in the rest of the foot, and in the fifth toe most of all. The cuticle of the second and third toes is wrinkled and beginning to separate. Sensation is entirely absent from the discoloured area. The superficial veins are unduly prominent

in left leg, and there is a slight amount of varix in the veins of both legs. The left leg is swollen, and is markedly cold, from the knee downwards. Pain is complained of on palpation over the upper part of the calf, and here there is slight induration. Pulsation is detected in the popliteal artery."

Three days after admission it was recorded that the discoloration extended to the ankle, the internal saphena vein was found to be corded immediately above the knee, and the induration of the calf was increased.

On July 31st she was seen by Dr. Alexander Robertson, who investigated her cardiac condition, and reported as follows: "Evidence of distension of *right* cavities of heart, with increased dulness to right. Evidence of distension of *left* ventricle upwards and outwards. Apex beat in fifth intercostal space half an inch external to nipple line, and three and a quarter inches to left of sternal margin. A distinct thrill is felt over the apex, and a pre-systolic mitral murmur (more widely diffused than usual) detected on auscultation. There is an accentuation of the second sound over the pulmonic area, but no reduplication. Cardiac sounds are nearly equal in intensity: short, sharp, and irregular all down the sternum. A faint systolic murmur occasionally heard in the aortic area, but not distinctly in the neck."

August 3rd. Right hemiplegia suddenly set in—face, arms, and leg, with inability to speak; eyes drawn to the left. The gangrenous area was still extending, the thigh was oedematous, and the arterial pulse could not be felt at the groin. Urine contained a considerable amount of albumen.

On August 8th it was noted that she was unable to understand what was said to her, and that the veins of left thigh were thrombosed as high as the saphenous opening. The pulse became very feeble, irregular, and rapid, respiration was irregular, but there was not marked dyspnoea. She slowly passed into a state of coma, and died early on the morning of August 9th.

Post-mortem examination made by Dr. Findlay, whose report follows: Dry gangrene of the toes of the left foot, the blackness extending to about the middle of the metatarsal

bones on dorsum and sole, and a dusky red blush extends from this upwards to about the middle of the leg, where the epidermis is raised in bullae. The discoloured area ends by an abrupt "line of demarcation," but there is no ulceration. Oedema of thigh with hardness, especially along posterior aspect.

Thorax.—About four ounces of clear, straw-coloured fluid in pericardium. *Heart* is considerably enlarged. Aortic and pulmonary valves are competent. Tricuspid opening is dilated and admits five fingers easily. The right side of the heart is dilated. The mitral curtains are much thickened, and have become adherent, forming a dense, thick, and rigid diaphragm in which is an aperture capable of admitting one finger only. There are thus conditions for obstruction and regurgitation. The valve is much thickened and cartilaginous from old endocarditis, and a few small old and fresh vegetations the size of a pin head are found on the auricular surface. The ventricle is dilated and much hypertrophied; the muscle is of good colour and consistency. The dilatation is most marked in the left auricle and especially in the appendix, which is occupied by a greyish laminated ante-mortem clot, which is very easily broken down. *Left lung* shows on the diaphragmatic surface slight fibrinous exudation, and near to this two small haemorrhagic infarctions, each with two free surfaces. *Right lung* presents six small haemorrhagic infarctions, varying in size from one inch to a little more than two inches square. The two largest areas are situated at the junction of costal and diaphragmatic surfaces; another is seen at the anterior border of the lowest lobe, and two others are seen, one in the middle lobe and one in the upper lobe. In all of them a white embolus can be detected in the small branch of the pulmonary artery supplying the area of the infarction, and the white clot is usually situated some considerable distance proximal to the lesion. Patches of fibrinous exudation exist over these haemorrhagic areas.

Liver.—In a nutmeg condition, with some cirrhosis.

Kidneys.—Left kidney shows in all five healed infarctions. One of these in the cortex is of a dark colour, the others are

situated, in the pyramids and have a caseated appearance. Right kidney shows one deep scar from old infarction. Both kidneys are small and are in a late stage of cirrhosis. Surface of the section very granular, and capsule removed with difficulty.

Spleen, not enlarged; shows one infarction of old standing.

Vessels.—The vena cava inferior contains a white thrombus which extends for about three inches from the junction of the common iliac veins, and is connected with thrombi in those veins, but is not adherent to the vessel wall. Left iliac vein is found completely occluded by a thrombus intimately connected with the wall and extending into femoral and saphena veins. Right iliac vein also thrombosed, and the clot extends into the femoral vein. Left femoral artery shows at its point of division a white thrombus completely occluding the artery, and behind this is a red thrombus. Before the white thrombus the artery is empty excepting for a few small white granular thrombi scattered up and down.

Head.—Arteries of the circle of Willis show slight atheromatous change, especially the right middle cerebral. On the left side the middle cerebral artery just after giving off its medullary branches is firmly occluded by a white clot. Externally, nothing remarkable can be detected in the motor area of the convolutions, but on section the grey matter of the ascending frontal and ascending parietal convolutions is found to be in a condition of yellow softening. The grey cortex is wanting in the normal appearance of layering; it is of a yellowish colour, and spreads out into the white centre without any distinct line of demarcation. The island of Reil is also in a condition of yellow softening, but the corpus striatum, optic thalamus, and internal and external capsules appear normal. No abnormality in the rest of the brain can be detected.

Commentary.—The conditions in this case are not difficult to understand. Old standing mitral disease with vegetations on the auricular surface of the valve resulted in embolism of one of the arteries of the left leg, probably the popliteal near to its bifurcation, producing gangrene. This was followed by

thrombosis of the veins of the affected area; and the progressive filling of the veins of the leg till not only they were completely blocked, but those of the right also began to be affected in the same manner. From the venous clots so formed portions were dislodged and carried into the circulation of the lungs producing pulmonary embolism. The infarctions were mostly in the right lung, were of small size, and widely separated, presenting in these respects a marked contrast to the conditions found in Case 1. As might have been expected, respiratory difficulty was not a pronounced feature of the case, and indeed was only noticeable within an hour of the fatal issue. The blocking of the middle cerebral artery and consequent hemiplegia may have been due to a small clot which had managed to pass through the capillaries of the lungs to the left heart, or to an embolus derived directly from the vegetations on the mitral valve or from the white thrombus in left auricle. The appearance of the clot suggested the latter origin rather than the former. The same remark applies to the infarctions in the kidneys and spleen. Several of these were old lesions, and must have commenced before the gangrene took place; if so, they probably were of cardiac origin, and it is likely that the more recent ones originated in the same way.

The patient was sent into the Infirmary with a view to amputation of the left leg, but the operation was never performed. We waited in vain for a limit to be set to the gangrenous process, and as we watched the case day by day found that the thrombosis of the veins was still progressing, till every vein was blocked as high as Poupart's ligament. On the other hand, she did not appreciably suffer from the presence of the gangrene; there was little or no pain, and no indication by rise of temperature or otherwise that any septic absorption was taking place. Her cardiac condition, also, was so bad and the pulse so feeble that the direct risk of operation was by no means small. Added to these things we had the onset of hemiplegia nine days after admission into hospital, and with this condition ended the chance of operative interference.

CASE 3.

Mrs. G., aged 50, charwoman. Admitted to Ward 26 on August 12th, 1899, having been run over by a tram-car. She was deeply under the influence of alcohol, and it was not possible to get a satisfactory history of the accident. There was a V-shaped lacerated wound on the anterior aspect of the middle third of the left leg, extending down to the muscles; also a small wound which extended into the subcutaneous tissue at the lower third of the leg. Another wound, of superficial character, was situated about half an inch above the left Poupart's ligament, running parallel to it. There was also a large abrasion over the epigastrium, a scalp wound over the occiput, and an ecchymosis of the right side of the abdomen. When examined by me on the morning after admission I was satisfied that there was a fracture of the pelvis, but the extent and nature of it were not satisfactorily determined. From the time of her admission the most prominent symptom was vomiting; the vomited matter was at first the contents of the stomach, then became biliary, and later had more or less of a faecal character. Nothing seemed to have the slightest effect in controlling it. There was some distension of the abdomen, and much difficulty in obtaining a movement of the bowels.

On the 14th August our attention was drawn to the question of obstruction, but before I had completed my ward visit the administration of an enema had brought away a large motion. On the following day (August 15th), however, the symptoms of obstruction continuing, it was considered advisable to put her under an anaesthetic and make further examination, and it was thought that abdominal exploration might be necessary. Under chloroform it was found that there was a separation through the symphysis pubis, and a fracture through the horizontal ramus of the pubic bone of the left side, as also a fracture through the ramus of the ischium and pubes. On examination *per vaginam* the fragment thus separated and depressed could be replaced, but became immediately displaced

when the fingers were removed. There was also found to be either a fracture into the great sciatic foramen or a separation of the sacro-iliac joint. No injury to urethra or rectum could be made out. When under the anaesthetic patient became bathed in a clammy sweat, and the pulse became extremely rapid and feeble. It was decided that no further exploration was possible. She gradually grew worse, and expired about eight o'clock the same evening.

Post-mortem examination. "*Heart.*—Aortic and pulmonary curtains are competent, and all the valvular structures are healthy. Aortic opening has a circumference of 110 mm., and tricuspid of 130 mm.

"*Right lung* is oedematous. A large ante-mortem clot, evidently the result of embolism, occupies the right pulmonary artery; it does not quite fill the lumen, but lies loosely in it.

"*Left lung* oedematous; no clots found in vessels.

"*Abdominal organs* healthy, excepting the kidneys, which are in an advanced stage of interstitial nephritis. There is general acute peritonitis.

"*Pelvis.*—There is complete rupture of the ligaments of the symphysis pubis. The horizontal ramus of the pubes is fractured on the left side, midway between the symphysis and pectineal eminence; there is also a fracture of the ischio-pubic ramus on that side. Another fracture splits the sacrum from above downwards, very close to the synchrondrosis; the second and third sacral spines are also crushed. The right femoral vein is thrombosed, and this has probably given rise to the pulmonary embolism."

Commentary.—This is a plain-sailing case. The tram-car evidently passed over the pelvis and crushed it; as a consequence of this crushing the femoral vein became thrombosed, and a portion of the clot was carried into the right pulmonary artery, and pulmonary embolism resulted. The symptoms of intestinal obstruction are less easily explained. The vomiting in the first twelve hours was regarded as the usual stomach upset consequent on a heavy drunken debauch, and probably this was the case. The distension of the abdomen and tenderness on pressure were due to the peritonitis, and this

was the result of a contusion of the abdomen, the evidence of which was the extensive abrasion over the epigastrium described.

No doubt the later evidences of obstruction were the result of the general peritonitis. On the day of patient's death she was removed to the operating theatre for more complete examination, and it was noticed that in lifting her from the bed to the "ambulance barrow" adequate care was not taken to keep the pelvis fixed, the result being that the pelvis dropped between the carriers of the trunk and legs. Despite the fact that a firm bandage was fixed round the pelvis, I have little doubt that harm was done in thus lifting her, and I am satisfied that the great displacement of the fractured pubic bone found on examination was due to this cause. Further, I think it probable that the displacement of the clot from the thrombosed iliac vein took place at the same time, for her condition grew rapidly worse from the time she was removed to the theatre. This case serves to emphasize the fact that extreme care is necessary in removing patients in regard to whom any suspicion of pelvic fracture exists.

EXAMPLES OF ABNORMALITIES OF THE PENILE RAPHÉ.

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Dispensary of Royal Hospital for Sick Children.

THE following cases have come under my notice during the past twelve months, and I have grouped them together under above title for obvious reasons.

In Case 1 the cyst did not, it is true, actually show a connection with the raphé, but the situation on the under surface of the prepuce is regarded by some as indicating the congenital nature of such tumours—the view being that they arise from vestigial epithelium at the time of closure of the lips of the uro-genital furrow to form the urethra. Again, the presence of a *stratum granulosum* shows the cyst to be of epidermic rather than of mucous origin, while the sebaceous contents point to this conclusion. The question of the nature and origin of these cystic formations has been already examined in my former papers^{1,2}, so that I need not say anything further here.

In Case 2 the condition of the scrotal raphé is as if closure had progressed almost to completion, and had then stopped. The furrow which is shown explains the above mentioned inclusion theory of cysts of the penis.

In Case 3 we are presented with a curious condition of the parts, inasmuch as a normally placed urethra is associated with the markedly devious raphé. Again, it is difficult to

¹ *Glasgow Medical Journal*, June, 1898.

² *Ibid.*, January, 1899.

explain the bifurcation of the anterior end of the raphé with an enclosed area of skin. In one of the papers mentioned above ⁽¹⁾ I quoted a case of Thöle's (*q.v.*), in which a slight deviation of the raphé to the right was accompanied by an elongated tubular cystic formation. This case was the subject of a degree of hypospadias comparable to what we have here, and Thöle explains the cyst and the hypospadias as concomitant evidences of antenatal errors in the development.

CASE 1.

Cyst occurring on the under surface of the prepuce.

I am indebted to Dr. T. K. Dalziel for the opportunity of examining this case. The patient, aged three years, was brought to the Western Infirmary on account of a swelling situated in the prepuce; an exact history as to the time of its appearance could not be obtained. The child was in all other respects healthy. The prepuce was very long and could not be retracted, and on its under surface at the anterior fold was situated a cyst of globular shape, measuring 6 mm. in diameter. The cyst was to the right of the middle line, while the raphé took a straight course forwards, passing over the mass well to the left of its equator (Fig. 1). The swelling was semitranslucent, of a yellowish tint, and was soft and semifluctuant. So far as could be determined the skin over it was not adherent, although thinner than normal. The genitals were otherwise normal. The parts were removed by circumcision, and were subsequently hardened in alcohol.

Examination after hardening showed the cyst to be lined by a smooth wall; the cavity was completely filled by a cheesy mass. Under the microscope the mass was seen to consist of desquamated flat epithelial cells, more or less in a condition of fatty degeneration. Here and there opaque granular masses were seen. These fractured on pressure being applied to the cover-slip, and were evidently composed of clumps of epithelial cells which had undergone extensive fatty degeneration. A portion of the cyst-wall was cut in

paraffin, the sections being made transversely to the long axis of the prepuce. Microscopic examination showed the structure to consist of several layers of flattened epithelium. Those next the cavity did not stain at all well, and in places these superficial layers were observed to be desquamating. The outer layers were not so markedly flattened, and their nuclei took on staining deeply. Here and there superficially were seen cells containing eleidin granules. The cuticle of the prepuce was separated from the cyst epithelium by a narrow layer of fibrous connective tissue, well nucleated. The epidermis, while here and there showing interpapillary down-growths, was on the whole flat, and did not present any glandular formation. In the sections examined no connection with the raphé was made out.

CASE 2.

Bilobate formation of the scrotal raphé, in a case of hypospadias.

The patient was aged 6 years, and was otherwise healthy and of normal appearance. The usual characters of a hypospadiac penis were well marked, especially the flexion of the organ (Fig. 2). On the ventral aspect of the glans was the meatal dimple, succeeded by a furrow which extended backwards to the urethral opening (Fig. 3), which was situated on the proximal half of the penis. At its anterior end, just behind the meatus, minute orifices were present in the deeper parts of the furrow. The raphé presented as a median ridge commencing at the root of the penis about 6 mm. behind the urethral opening. Traced backwards on to the scrotum it was found, at a distance of 7 mm. from its commencement, to be formed of two lips with an intervening furrow, and this arrangement extended to the anterior end of the perineal portion, where it assumed the normal appearance of a median ridge. The bifid arrangement of the structure stopped suddenly, where it joined the median ridge on the upper and anterior part of the scrotum, the lips appearing to balloon out so as to form each a small but distinctly observable swelling. These

swellings did not appear, on palpation, to be anything beyond a hypertrophic condition of the skin folds, although at first sight they resembled cystic formations. I am indebted to Dr. Hector Cameron for permission to examine this case and to have the drawings (Figs. 2 and 3) made.

CASE 3.

Abnormal course of the penile raphé, in a case of slight hypospadias.

The patient, aged 7 years, was apparently healthy otherwise. He presented a slight degree of urethral malformation, the opening being situated just behind the margin of the glans. He attended at the dispensary of the Western Infirmary on account of some difficulty in micturition, due to narrowing of the abnormally situated urethral orifice. This was treated by incision, and he has not had any repetition of his troubles.

The glans was ventrally flexed, as if bridled. There was a well-marked meatal depression which tapered posteriorly, this extremity being very slightly overhung by the somewhat elevated commencement of the urethral groove; this elevation formed a distinct barrier between the depression on the glans and the actual opening of the urethra. The under surface of the penis presented no evidence of raphé, the skin in this situation being perfectly smooth and white. Further inspection showed that the scrotal raphé passed forwards in the normal manner on to the root of the penis, but the penile raphé almost immediately took a sweep up on to the right side of the organ. Traced forwards it followed a wavy course and ended on the right side of the hooded prepuce near the origin of the latter from the skin of the penis. At this point it became slightly thickened, and then bifurcated into dorsal and ventral portions. The latter coursed directly inwards to end at the opening of the urethra, while the dorsal reached the same point after running forwards and downwards along the folded outer surface of the prepuce. Each of these divisions was of slighter proportions than the rest of the raphé, which presented the appearance of an undulating cord, the brown

colour of which contrasted markedly with the otherwise pale integument of the penis and scrotum. On introducing a probe, the ur  thra was found to run in the usual situation in the corpus spongiosum, *i.e.* in the middle line.

EXPLANATION OF PLATE.

Fig. 1.—Ventral view of the penis of Case 1, showing the relation of the cyst to the raph  .

Fig. 2.—Lateral view of parts in Case 2, showing “bridling” of the penis.

Fig. 3.—Ventral view of the same, showing the abnormal appearance of the raph  .

Fig. 4.—Lateral view of Case 3, showing abnormal course of penile raph  , with slight “bridling” of hypospadi   glans.

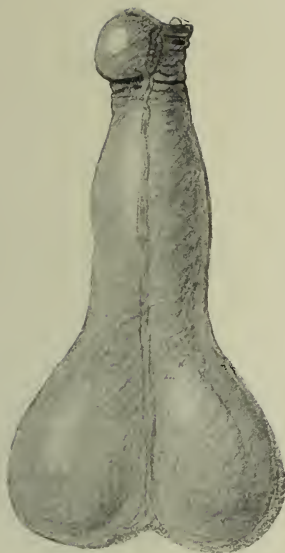


FIG. 1.

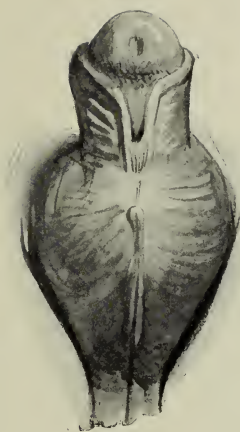


FIG. 3.



FIG. 2.



FIG. 4.

THE OPERATIVE TREATMENT OF SPINA BIFIDA AND HYDROCEPHALUS.

By JAMES H. NICOLL, M.B.,

Assistant Surgeon, Western Infirmary; Extra Surgeon, Children's Hospital; Consulting Surgeon, Ear Hospital; Consulting Surgeon, Glasgow District Lunacy Board, etc.

A YEAR ago I read before the section of Diseases of Children at the meeting of the British Medical Association in Edinburgh a paper on the "Radical Cure of Spina Bifida," based on an experience of thirty-two cases which I had treated up to that time. That paper was published in the *British Medical Journal* for October 15th, 1898. It was subsequently, on the request of the editor of that journal, published in *Pediatrics*, and reproduced in brief in the *Archives of Pediatrics*, and it has since been quoted by several writers.

During the year which has intervened since the paper was published, I have had a number of letters from professional brethren requesting advice on cases of Spina Bifida and Hydrocephalus, and have had referred to me twenty-one further cases of these affections. Of these cases several presented features of sufficiently exceptional interest to warrant their inclusion in such a publication as the *Glasgow Hospital Reports*. And, in view of the publicity which has been given to my latest paper, it may be well to take this opportunity of summarizing what I have at various times published on the subject in the *Glasgow Medical Journal*, the *British Medical Journal*, and the transactions of several societies.

The conclusions which the results so far have led me to may be stated as follows:

That treatment by injection, whatever its merits in times past, is, in comparison with the open operations which modern aseptic practice has made possible, uncertain in its results, of high mortality, and unsurgical in conception.

That open operation, in suitable cases, has a mortality little, if at all, higher than has the operation for the radical cure of inguinal hernia. That this does not apply to cases where, on account of difficulty over nerve tissues, incomplete or partial operations have to be adopted. That the term "suitable case" applies to cases in which the sac is not ruptured, ulcerating, or sloughing, and in which there is not present an extreme degree of hydrocephalus. The presence of such conditions (*vide British Medical Journal*, October 15th, 1898), while not constituting a bar to operation, rather, perhaps, rendering the need for operation the more urgent, greatly increases the chances of the operation failing to save the life of the child.

That open operation in cases in which the conditions permit of a complete operation accomplishes a "radical cure" of spina bifida. Out of forty-six cases operated on, I have so far had only three cases in which, after complete operation, recurrence took place. Two of these were subsequently remedied by second operation.

That the radical cure of spina bifida proves in some cases a remedy for the accompanying paralysis of lower limbs, bladder, and rectum. In several cases which I have had the removal of bands of spinal cord or nerves from positions of tension, stretched over the walls of the sac, and the replacing of them in the spinal canal, have resulted in relief of the paralysis—partial in some cases, complete in others.

That in cases where hydrocephalus accompanies spina bifida the operative measures adopted for the latter may be made the means of treating the former, or combined with measures to that end. I have treated a number of cases of hydrocephalus by one or other of the methods described below. What opinion is to be formed of the

PLATE I.

(Plate IV. in publication quoted.)



Facing p. 298.

ultimate results can only be determined after a period of years. At present these cases are under observation, and in some of them a marked relief has followed treatment, while in several, cure appears to have resulted. In all cases, however, I should wish to wait until puberty is past before expressing too definite an opinion.

The methods of operating I have followed have been fully described elsewhere (*British Medical Journal*, October 15th, 1898). They have, for the most part, included the use of flaps of skin dissected from the base of the tumour, with, in addition in many cases, flaps of mesoblastic tissues (fascia, muscle, and even bone) raised from the margins of the gap in the spinal canal.

They vary chiefly in the manner of dealing with the sac of spinal membranes, and may be briefly stated as follows:

(a) Pure meningoceles, without contained nerve cords, are simply cut away, and the neck closed by either ligature or suture, and covered by the flaps.

(b) Meningoceles which contain nerve tissue—either spinal cord or spinal nerves: in such cases I have employed two different methods. One of these would appear to be in some respects new. Since I first published it it has been quoted with my name attached (*vide* the recently published text-book of *Surgery* by Pick). That being so, it may be well to reproduce here the last published account of it. The following extract, with two accompanying photographs, is taken from the paper of October 15th, in the *British Medical Journal*:

“Cases in which the sac on being freed and opened is found to contain nerve cords.”

“In my earlier cases, several of which were shown on different dates to the Glasgow Medico-Chirurgical Society, I followed the practice of dissecting the cords off the interior of the sac. This is not free from risk, and in one case, operated on in 1895, I produced damage in detaching the

nerves which caused paralysis of one leg, which has remained permanent.

“To avoid such risk I adopted a plan which, in connection with the demonstration of several cases of cured spina bifida, I described to the Glasgow Medico-Chirurgical Society in November, 1897. The method is as follows:

“Flaps of skin and mesoblastic tissues are dissected up in the usual way. The sac is freed and opened. Such portions of it as are free from nerve tissue are excised. The remainder is ‘cut into ribbons’—by incisions made from the interior, parallel with the nerve cords incorporated with it—and thoroughly roughened with the point of knife. The ‘slashed’ and roughened sac is then placed in the patent spinal canal, over which the flaps are sutured in the usual way.

“In a certain number of cases—two of which have been mentioned above, and two of which (Plates IV. and V.) are described below—I have still further extended the principle and considerably modified the method of this operation, with good results.

“Plate IV.: M. D., shown to the Glasgow Medico-Chirurgical Society in April, 1898, was sent to me, at the age of 5, in 1896, by Dr. Lamont, of Chryston.

“*Particulars of case.*—Spina bifida tumour 8 by 9 inches across. Complete paraplegia; constant incontinence of urine; condition of bowel in which obstinate constipation alternated with incontinence of faeces and flatus.

“At that time I operated—opened sac by incision shown in plate—found it so occupied by expanded nerve cords that excision of even small portions appeared impossible. With the knife I carefully tore such small areas as seemed free from nerve tissue, and gently, but pretty thoroughly, roughened the whole interior, including the surface of the nerve cords in many parts, and stitched up.

“Condition when shown in April, 1898, nearly two years after operation:—Sac replaced by a tough lobulated fibrous mass, shown in plate. This is considerably smaller than the pre-existing sac, measuring 4 inches by $3\frac{1}{2}$ inches

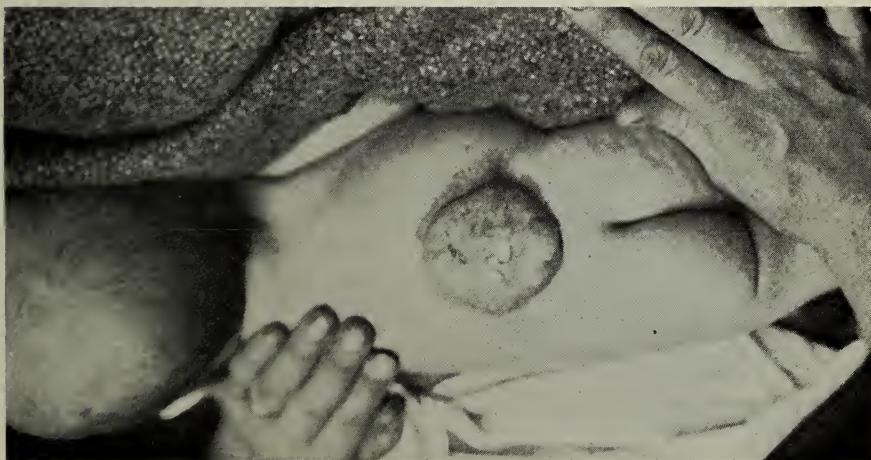
(Plate II. in publication quoted.)



EXCISION OF SPINA BIFIDA SAC.

Scar of wound eight weeks after operation, showing puckered elevated ridge produced by method of suturing. Ridge already largely obliterated.

(Plate V. in publication quoted.)



across. It is not tender, is not altered in size by pressure, and was compared in consistence to a lipoma or a fibroma by various members of the Society who examined it. The child can walk several miles at a time, and has nearly perfect sensation in the legs, which are, however, in some respects, like the limbs of infantile paralysis, being bluish and distinctly deficient in muscular bulk. The bowel acts regularly, and the sphincter is now efficient. Urinary incontinence has completely ceased except that, as the mother states, a little involuntary escape is apt to occur if the child is made nervous or excited.

"Plate V.: Infant at present in the M'Alpin Nursing Home. The plate shows a hard fibrous mass, unaffected by pressure, occupying the site of a former spina bifida tumour of nearly thrice its size.

"The sac was opened five weeks ago by a median incision, and was found largely covered by nerve cords. Its interior was roughened in the manner above described, and closed by suture.

"It is now practically obliterated. My reason for breaking the rule I have laid down for myself, and producing this case at this early date after operation, is that, as will be gathered from the plate, the case illustrates what I believe to be a fact—viz., that, unless present in an extreme degree, hydrocephalus constitutes no bar to successful operation on spina bifida. The plate indicates what is the fact, that the patient suffers from very pronounced hydrocephalus.

"At the meeting in November, 1897, when I first described this method of operating, I stated that, in searching the literature of the subject, I had found a paper by Mayo Robson, of Leeds, in the *Annals of Surgery* for July, 1895, in which most interesting communication Professor Robson says:—'In cases where the cord is expanded, or nerves are blended with the sac, excision of redundant parts or incisions between manifest portions of nervous structures reduces the tumour, and enables it to be placed in the canal, and usually there is no difficulty in covering the replaced structures.'

"The method, therefore, into which my experience has

gradually led me would seem to be in some respects a combination of the principles of Professor Mayo Robson's operation for spina bifida and Professor Macéwen's operation for aneurism.

"It differs from Professor Robson's in that no attempt is made to reduce the bulk of the tumour by the operation, that end being attained by the fibrous contraction and consolidation which follow; and, further, in that operative procedures are not confined to the portions of the sac between the nerve elements; and, finally, that in certain cases (viz., such cases as Plate I., where the sac was torn and roughened internally by a needle passed into it through the skin) the procedure has no similarity whatever with Professor Robson's operation."

Since the foregoing was published I have for certain cases (and certain cases only) adopted a modification of the operation. The "slashed" and roughened sac is gently packed with sterilized iodoform or other gauze and partially sutured. At the end of twenty-four to forty-eight hours the packing is removed and the operation completed by suture of the flaps in the usual way. It is perhaps not necessary to remark that such a method of operating is, equally with such drainage as was carried out in the case of Baby B. (*Multilocular lateral spinal cyst*), given below, only justifiable under a rigid system of special nursing. The child lies prone on a pillow on the nurse's knees for a week or longer. During that time the back is constantly exposed to view so that contamination of the dressing by urine or faeces is impossible.

It will be evident that the local result of such an operation must be less satisfactory than the result in cases where the conditions permit of a complete excision of the sac, as in Photographs II. and III. reproduced from the same paper.

In the treatment of hydrocephalus I have employed several methods described in the paper quoted. Of their comparative values I have not yet been able to form an

PLATE IV.

(Plate III. in publication quoted.)



EXCISION OF SPINA BIFIDA SAC.

Scar of wound three years after operation—position of extremities indicated by pencil marks. Puckering completely obliterated and scar very faintly seen.

Facing p. 302.

opinion sufficiently decided for publication. They are, briefly, the following:

(a) Drainage of cerebrospinal fluid at the time of operating for spina bifida. Into the vexed question of the route or routes by which hydrocephalic fluid reaches the sac of a spina bifida it is not necessary to enter here. It is sufficient to re-state the fact that in certain cases of spina bifida with hydrocephalus it is possible, by raising the child's head and shoulders after the spina bifida sac has been opened, to drain away as much fluid as may be deemed safe, the amount being judged by the tension of the fontanelle.

(b) Continuous drainage by a tube inserted into the spinal canal through the neck of an excised spina bifida sac at the time of operation. In several cases I have kept such a tube draining fluid into the dressings for a week.

(c) The establishing of a drainage channel from the spinal canal (at the site of, and during the operation for, spina bifida) into either the peritoneal cavity or the cellular subcutaneous tissue or both. This may be done by a drain which is absorbable after a time, or by a drain which is removed by secondary operation after a period of weeks or months.

(d) Drainage of the cerebral ventricles, either externally or into the meninges.

(e) Application of various solutions (chiefly iodine) to the interior of the cerebral ventricles. What experience I have had of this method has impressed me favourably. It is not, however, devoid of risk. The amount and the strength of an injection will depend on the degree of dilution likely to occur internally, that is, on what the operator knows, from previous drainage or otherwise, of the amount of fluid present. In any case, for a first injection something less potent than Morton's fluid should be employed.

Of the cases of spina bifida and hydrocephalus I have seen during the past year, some twenty-one in number, those following presented features somewhat out of the usual:

Case treated throughout as an out-patient.

In the paper in the *British Medical Journal* I stated, "It may fairly be said that in cases of spina bifida situated in the lower lumbar region the success of the operation is as much in the nurse's as in the surgeon's hands. The operation itself in cases of pure meningocele is of the simplest; and, when the tumour was placed in the dorsal or cervical region I have more than once operated on cases in the out-patient department, and had them taken home and nursed by the mothers with complete success." This case proves that spina bifida sacs situated in the lumbar region may be similarly treated.

Baby D., at the age of two months, was sent to me at the Children's Hospital on January 6th last by Dr. J. Scanlan. It had a spina bifida sac in the lower lumbar region about half as large again as a duck's egg. This I excised. Sister Laura undertook the dressing, instructed the mother as to the nursing, and visited the child regularly. The result is as the photograph shows (Plate V.). Union occurred so perfectly that six months later, the date of the photograph, the scar of the operation was practically imperceptible. The patient was shown to the Glasgow Medico-Chirurgical Society on April 21st.

Case of multilocular lateral spinal cyst.

Baby B., aet. six months, sent to me in October last by Dr. R. Crawford. The photograph (Plate VI.) indicates the size of the cystic tumour, but, owing to the position of the child, fails to show the site clearly. The swelling occupied an area corresponding with the right half of the sacrum, right sacroiliac synchondrosis, and a large part of the right ilium. It was elastic, had a perceptible respiratory wave, and became tense on exertion. There was distinct hydrocephalus.

The child was admitted to the M'Alpin Nursing Home for operation. I made a free incision from the middle line outwards and downwards towards the great trochanter, and partially isolated the cyst from the surrounding

PLATE V.



Case of Excision of Lumbar Spina Bifida which was treated throughout as an out-patient. Photograph six months after operation.

tissues. Finding isolation difficult, I incised the tumour and explored it internally. It consisted of a multiloculated sac, containing cerebrospinal fluid, and sending processes amongst the deep tissues. Crossing one portion of its space were four nerve cords, apparently going to form the great sciatic nerve. Just above this was a deep process of the cyst, and the finger passed into this seemed to go through the bone in the region of the sacroiliac synchondrosis into a recess extending into the retroperitoneal tissue. It being clear from the position of the nerve cords mentioned that any attempt at excision of this complicated and deeply-placed sac would involve the risk of paralysis of the limb, I abandoned operation, and closed the wound.

Three days later, the child progressing well, I removed one of the skin sutures and passed a rubber tube into the sac, securing it to the skin by suture. For a week that was allowed to drain. At the end of that time the tumour was much reduced in size, the fontanelle depressed (and pulseless, when the head was raised), the face shrunken, and the eyes "sunk" in the orbits. The tube was withdrawn, and another suture inserted. The child recovered perfectly, and remained well, and in April it was shown to the Glasgow Medico-Chirurgical Society. In July the child continued well, and the hydrocephalus was distinctly less marked. The cystic tumour, while of somewhat less bulk than formerly, and appreciably solid in the region of the scar, was unaltered. On July 3rd I punctured it with a trocar and cannula, evacuated some drams of fluid, and injected one dram of Morton's fluid. The result was severe illness of the child, marked by a period of over a week of high temperature and general twitchings; and at the present time, a month later, Dr. Crawford reports that the child is just regaining its usual health. The injection has, as yet, had little if any local effect on the cyst.

It need hardly be remarked that any such drainage as was carried out in this case is only permissible under nursing arrangements which are absolutely reliable; and I should wish to express my indebtedness to the nurses of the M'Alpin

Nursing Home, and to others, for the care with which they have nursed many of these cases. It is no light task to nurse a fretful infant night and day on its face for a fortnight, without once turning it round or permitting it to raise its shoulders, and without allowing the least soiling of the lumbo-sacral region. And when to this, in some cases, is added the presence of a constantly trickling drainage tube, the amount of flow from which is regulated from time to time by the relative heights of head and pelvis, the task becomes one of some little delicacy.

In Kirmisson's *Maladies Chirurgicales d'Origine Congénitale* is the record of a lateral cystic spinal tumour in the same region.

Cases in which the termination of the spinal cord was excised with the sac.

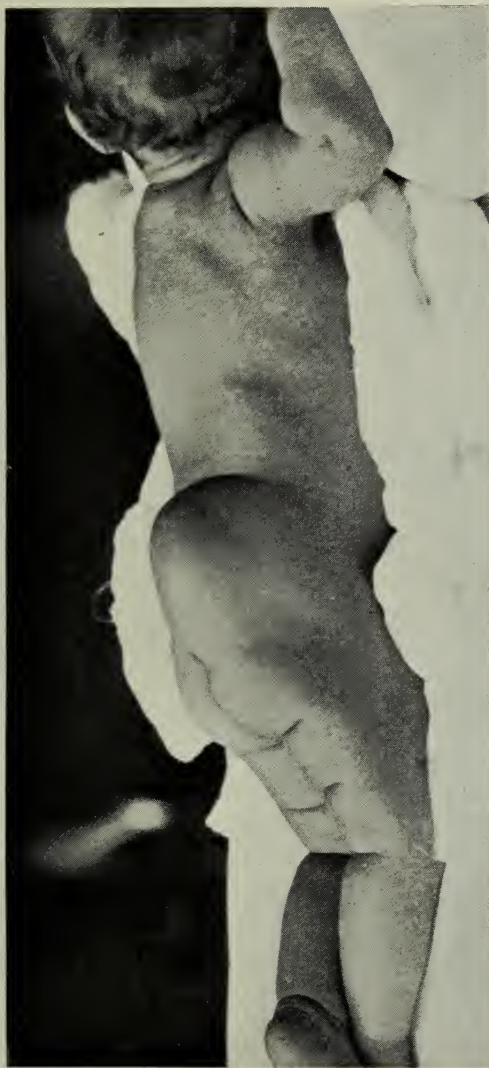
In spina bifida sacs situated in the lumbo-sacral region it is not uncommon to find a dimple produced by attachment to the dome of the sac of the terminal part of the spinal cord. In certain cases I have, after carefully isolating and displacing all accompanying nerves (constituting the cauda equina), excised this terminal attachment of the cord with the sac. This greatly simplifies the operation. I have seen no resulting paralysis.

Case 1.—Baby A., puny and feeble, sent to me, at the age of 2 months, in May last by Dr. Jas. Rutherford, with a spina bifida in the lumbar region nearly as large as an average orange.

This I removed in the Western Infirmary. The child made a good recovery. Plate VII. is a photograph of the dome of the sac, with attached to it $1\frac{7}{8}$ inches of the terminal central strand of the cord. Part of this has been removed and subjected to microscopical examination. It appears to consist solely of fibrous tissue.

The ellipse of sac and skin fused which is shown in the photograph represents a portion only of what was removed. After the flaps outlined by the elliptical incision had been

PLATE VI.



MULTILOCCULAR LATERAL SPINAL CYST.
(Case of Baby B.)

raised, and the sac isolated to its neck and opened, the remainder was removed in the usual way.

Plate VIII. is a photograph of the child taken six weeks after operation. The operation scar, as in the case of Baby D. (*vide* Plate V.), was so faint as to be all but imperceptible in the photograph. The negative was therefore "touched" in order to emphasize the impression. The result has not been happy, the impression given being that of a large irregular scar.

The child at present, three months after operation, is still decidedly feeble, as before operation. It has grown, however, and there is no indication of any paralysis.

Case 2.—Baby B., aet. 3 weeks, sent to me by Dr. Thomson, of Airdrie, in January, 1899, with a spina bifida in the lower lumbar region of the size of a Normandy pippin. This I excised in the usual way, and with it the terminal central strand of the spinal cord.

On the morning of the operation the child was found to be suffering from marked carbolic acid poisoning. Its urine was blackish-green when passed, and the child was markedly collapsed, and vomited frequently. In three cases I have seen similar symptoms follow the application of carbolic dressings to spina bifida sacs prior to operation; and it seems not improbable that the very thin parietes, bathed on one side by serous fluid, offer conditions under which absorption from the outside may occur with great facility. On the two former occasions on which symptoms of carbolic poisoning were present, I deferred operation. On this occasion the child appeared so ill that I thought it better to proceed with the operation in the hope that the evacuation of the contents of the sac might lead to relief of the symptoms. Unfortunately, this did not occur. The symptoms persisted, and were aggravated by increase of the vomiting and diarrhoea, with evidence of acute gastrointestinal irritation, accompanied by subnormal temperatures. The wound, in spite of the child's increasing weakness, healed before death occurred from exhaustion on the eighth day. During the period subsequent to operation there was no evidence of paralysis. The terminal

strand of the spinal cord which was removed was examined by Dr. Fullerton, who assisted at the operation, and myself, and is at present in my possession. It consists of a fine tube, with thick fibrous walls, covered by glistening endothelium. It is less than $\frac{1}{8}$ inch thick and about $1\frac{3}{4}$ inch long. It appears to be the filum terminale little, if at all, altered.

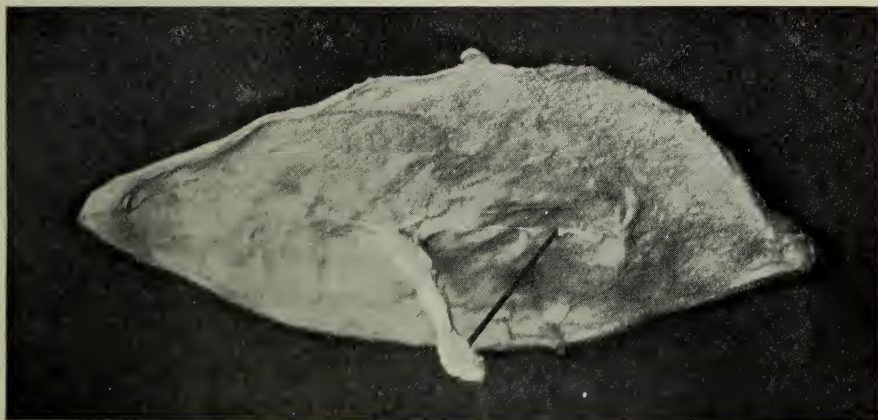
Case 3.—Baby B., sent to me, at the age of five weeks, by Dr. Lamont, with a spina bifida in the lumbar region of the size of a large Tangerine orange. This I removed in the M'Alpin Nursing Home in May, 1898, and with it a portion of the terminal strand of the cord measuring $1\frac{1}{4}$ inch. The child is at the present time well, and exhibits no evidence of paralysis. The part removed consists of nerve fibres and fibrous tissue.

*Cases of spina bifida complicated by abnormal patency
of the neurenteric canal.*

Such cases are recorded as rarities from time to time in surgical literature. An illustration of one is included in Vol. II. of Treves's *System of Surgery*. The records of the two following cases are given for what they may be worth in the unfortunate absence of anatomical demonstration of the existence of a patent neurenteric canal.

Case 1.—Baby H. was brought, aged two days, to me at the Children's Hospital on May 21st, 1897. The infant was "shrivelled" and puny. The anus was all but quite imperforate, being represented by an aperture which just admitted an ordinary surgical probe. This aperture was situated on the summit of a prominent swelling, which occupied the perineal region and ischiorectal fossae, and formed the projecting portion of a mass which occupied the true pelvis, and could be felt above the pubis. In the lumbar region was a swelling which presented all the characters of a spina bifida which had ruptured by ulceration, and through the rupture was discharging a fluid which in odour, colour, and consistence was clearly liquid faeces.

I dilated the constricted anal aperture till it admitted



Dome of sac excised with terminal central strand (fibrous) of spinal cord attached.

PLATE IX.



Photograph of Baby H.'s back, viewed from below, showing (1) Ulcerating spina bifida through which faecal material escaped; (2) Anal aperture, lacerated by previous dilatation; (3) Left leg pulled up under pelvis, showing wrinkles of loose skin due to emaciation.

my forefinger and gave vent to a quantity of semi-solid faeces. The child was sent home in the expectation that death would speedily ensue. In spite of its extreme feebleness, however, it lived for a week. During that time the faecal discharge from the lumbar orifice gradually ceased—though not entirely. Three days before death the photograph represented in Plate IX. was taken. The perineal swelling, which was evidently due to retention of intestinal matters, had by this time disappeared, the retained matters having found exit through the dilated anal orifice.

After considerable trouble permission was obtained for a post-mortem examination. This Dr. Primrose and I carried out. By means of free incisions we removed the entire lumbar spinal column with the sacrum in one piece, and, attached to this, all pelvic and abdominal structures in relation to it, including the rectum. That the swelling in the lumbar region was a spina bifida was evident in the deficiency of the laminae and spines. The specimen I put away in alcohol for dissection later. Most unfortunately it has been lost, having apparently disappeared in the course of removal of my household furniture to my present address.

Case 2.—On April 20th, 1899, I received from Dr. McLean, of Stonehouse, the following letter:

“I have a case here which will interest you. The child was born yesterday morning, and seems a healthy child, but has what I took to be a burst spina bifida in the lower lumbar region. When I was at the house to-day I was told that the faeces were ‘coming by the back.’”

I arranged accommodation for the infant in the McAlpin Nursing Home; but, as the weather was cold, it was deemed advisable to wait for a suitable day for the journey.

On April 28th Dr. McLean informed me that the child had died that day after a series of general convulsive seizures.

With a view to securing a post-mortem examination and a photograph, I at once communicated with Dr. McLean. To that end he used every endeavour. The parents, however, naturally enough, absolutely declined; and refused even to

permit me to see the body. Most unfortunately, therefore, an actual pictorial representation of the case is lacking.

At my request Dr. McLean put on record the facts of the case and sent his notes to me next day. These I reproduce here :

"The spina bifida occupied the lower lumbar region. Its size was that of a large hen's egg. It had originally evidently been larger; for it was ruptured, and clear fluid was oozing out, while the dome of the swelling was shrunk. At the neck of the sac was the orifice of a passage, large enough to admit my little finger, and surrounded by large red granulations. Through this passage the faeces were passed. The anus appeared normal, and the child had no other visible defect."

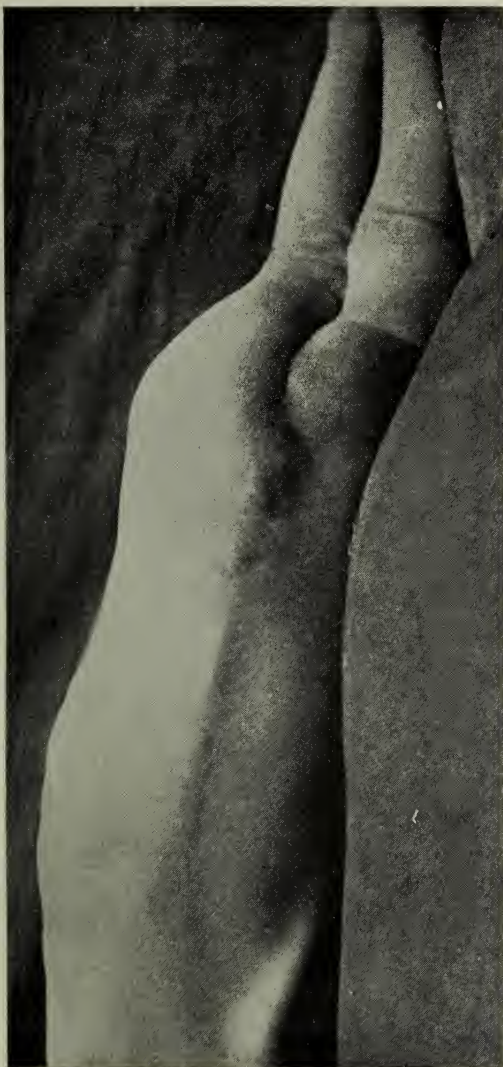
Case of cystic tumour (of patent neurenteric canal?).

B. T. was sent, at the age of three months, to me by Dr. R. Morton, on October 14th, 1898. Fig. X. represents the lower limbs and perineum of the child at that time. The cystic tumour depicted occupied a site between the anus below and the coccyx above. The coccyx was tilted backwards and upwards.

I excised the greater part of the mass. The specimen consists largely of firm fibrolipomatous tissue. It contains two cysts of the size of pigeon's eggs. One of these contained thin pultaceous material, which escaped during the operation. The other contained serous fluid. The wound healed partially, complete union being prevented by the constant escape of a clear thin fluid having all the characters of cerebrospinal fluid and the intermittent escape of bells of gas with unmistakable faecal odour.

At the end of three weeks I reopened the partially healed wound, and, dissecting more deeply, removed another cyst with thin walls and turbid serous contents. This, and the specimen formerly removed, were shown to the Glasgow Medico-Chirurgical Society on April 21st, 1899. Both are in my possession. At the end of seven weeks the wound had

PLATE VIII.



Case of Baby A.



contracted to a fistulous aperture, and the child was dismissed from the nursing home. It was shown to the Medico-Chirurgical Society on the date mentioned. At present the child is in good health, though distinctly hydrocephalic. The fistulous tract below the coccyx persists. No gas escapes now, but there is a frequently recurring flow of thin serous fluid in small amount.

The exact nature of this case is not quite clear. My impression is that the tumour was a multilocular cyst taking origin in a partially obliterated neurenteric canal, and that the operation opened the tubular communications with both spinal canal and rectum.

OPERATIONS FOR SQUINT BY ADVANCEMENT.
BEING SUBSTANCE OF A LANTERN DEMONSTRATION TO THE
MEDICO-CHIRURGICAL SOCIETY OF GLASGOW.

By FREELAND FERGUS, M.D., F.R.S.E.,

Surgeon, Glasgow Eye Infirmary ; formerly Ophthalmic Surgeon, Glasgow Royal Infirmary.

IN a communication made some time ago to the Medico-Chirurgical Society of Glasgow (see *Glasgow Medical Journal*, September, 1898) I endeavoured to point out the many advantages of muscular advancement over tenotomy in the treatment of all forms of strabismus. The cases there chiefly discussed were those of concomitant convergent strabismus, and the objections advanced to tenotomies in the treatment of this affection were as follows :

1. Concomitant convergent strabismus in the vast majority of cases can be shown to be associated with a defective action of the external recti, and not with an excessive action of the internal recti. If this be so, it is more rational to attack what is defective than to interfere with a function which is not impaired, to strengthen the power of relative divergence without impairing the convergence.

2. By performing advancement we are the more likely to restore binocular vision. The ideal end of all operations for strabismus is the restoration of binocular fixation. No doubt in many cases this ideal cannot be attained, and the most that sometimes can be expected is the aesthetic effect of the reduction of an obvious deformity.

3. Tenotomy very often causes deformity. Thus generally after a tenotomy, the inner canthus will be found to be widely

dilated, and the movement of the eyeball in the direction of the divided muscle much impaired, while along with these changes there is a certain amount of exophthalmos.

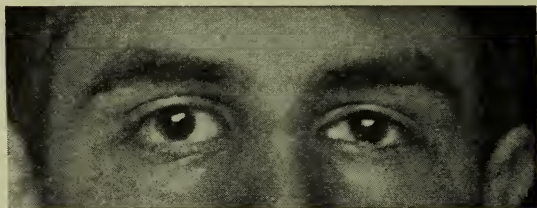
Occasionally also after tenotomy there is marked divergence. Particularly is this true when the operation has been done during the administration of a general anaesthetic. It occurs in a very considerable percentage of cases, and in the hands of even the best operators. A hideous divergent squint after tenotomy is not always the result of bad operating, although it is often supposed to be so, but in many cases is a natural sequence of the operation selected.

4. In many cases the squint is not apparently diminished by the tenotomy. No doubt accurate and careful measurements would reveal a certain diminution in the angle of the squint, but the disfigurement caused by the squint is not appreciably lessened. Such were the objections which I advanced in the paper referred to against tenotomy for squint.

It is my present purpose to give a few diagrams of certain cases before and after operation. These have, possibly with one exception, been made from negatives which I have personally taken. At first only an ordinary camera was used, but more recently I have employed the apparatus devised by Maddox.

It is impossible to teach any part of surgery clinically by diagrams, and no competent teacher will attempt it. What would be thought of a surgeon who, teaching clinically, instead of showing actual cases and operations, contented himself with mere pictorial representations? Hence I deeply regret that in the present communication I have to fall back on so crude a method as the exhibition of photographs. It is impossible that by the inspection of these anything like clear information can be obtained. It is impossible on a lantern slide to measure fields of fixation or the range of the convergence. Moreover, a picture can never reproduce nature. As thrown on the screen, however, these pictures have one advantage; they are considerably magnified, so any defects are more easily seen and good results more readily identified.

Before any absolute conclusion can be drawn as to the eyes being placed bilaterally symmetrically, it is necessary to have the patient before us for examination. The corneal reflex as seen in pictures is no doubt a very fair criterion, but it is not an absolute one, in so far as it gives no indication whatever as to the axis of vision. It is, however, the only one available for my present purpose.



E. H.

The first picture is that of a lad, E. H., aged about 18. The present negative was made only a few days ago. Eight years ago, that is when the patient was aged ten, I performed a tenotomy of his right internal rectus. The operation was done under chloroform. Two obvious defects at once strike the observer. (*a*) The inner canthus of the right eye is found to be extremely wide. Measured on the picture it is found to be about one-third as wide again as the other canthus. Hence there has been produced an unsightly deformity.

(*b*) It is apparent from the picture that the right eye is rotated slightly out or the left slightly in. The position of the patient at the time of being photographed was distant fixation as nearly as possible in the line of the camera lens. The left eye, being the better, is the one used for fixation, and the right is allowed to assume its natural position—in this case one of actual divergence. The distance between the inner margin of the right cornea and its own reflex is less than the corresponding distance on the other eye. Moreover, the distance between the inner margin of the cornea and the inner canthus is appreciably greater on the right side than on the left. There is, as a matter of fact, a divergence of several degrees as measured on the patient.

The condition of the patient at present is,

Right Sph. +3, cyl. +1, axis horizontal, V.A. = $\frac{20}{60}$.

Left Sph. +4, V.A. = $\frac{20}{20}$.

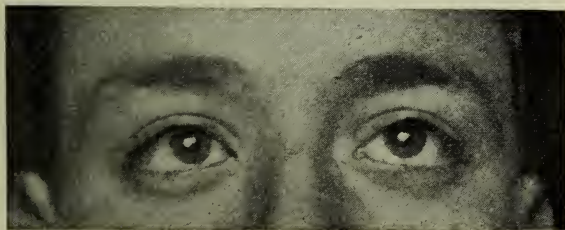
It may, of course, be said that this proves nothing except that the operation was bad. As regards the operation itself, it was a simple tenotomy, the division being restricted as much as possible to the tendon of the muscle. Dilatation of the inner canthus is even in the hands of the most celebrated operators a not infrequent occurrence, and none of the numerous procedures which have been invented for its prevention seem to be of the slightest avail.

As regards the divergence, that is a condition which I have not seen following any amount of advancement for the cure of strabismus convergens except in one case in which tenotomy had previously been performed. This case has already been mentioned in my previous paper.

A result such as has been obtained here renders binocular fixation, to say nothing of binocular vision, impossible, and although it lessens the unsightliness of the original condition still leaves much to be desired from an aesthetic point of view.

On proposing to operate for convergent squint, a fear is often expressed that after the operation the patient will squint outwards. This is a popular opinion, but not a delusion, for with tenotomy it frequently happens.

The second case which is shown is one in which the operation by tenotomy has not removed the defect.



J. P.

J. P., presently aged 17, was subjected to the operation of tenotomy of the right internal rectus fully four years ago. This photograph was taken after the operation with the

patient directly facing the camera, with distant fixation as nearly as possible in the axis of the lens of the camera.

On careful examination it is found that there are still 15° of convergent strabismus as measured on the perimeter arc. That, of course, is perhaps not the actual amount of squint, but is approximately correct. The patient was found to be so amblyopic that the attempt to measure the true amount of squint by means of prisms or by projection had to be abandoned.

The following notes were made in this case :

Vision of right eye = fingers counted at 18 inches.

Vision of left = $\frac{2}{20}$.

Right eye—hypermetropia of about 2.5 D with astigmatism of 1.5 D against the rule. These figures were estimated with the ophthalmoscope and ophthalmometer.

Left eye—nearly emmetropic.

The fundus in each eye looks perfectly healthy except that in the right the vessels are slightly tortuous. On examining the photograph it will be seen that the right eye is much nearer the right internal canthus than the left eye is to the corresponding canthus. The corneal reflex of the left eye is in front of the pupil, while that of the right is displaced slightly outwards—more of the pupil is seen.

These two cases typify the worst defects of tenotomy ; in one there is divergence, in the other convergence. Both no doubt can easily be remedied by advancement of an internal and an external rectus respectively. In the case of E. H. it is probably advisable, for there is a fair amount of vision in each eye, but the other case may be left alone, for the result at present has so much improved the apparent defect as to give satisfaction to those who examine the patient only casually.

The next case, Miss P. L., aet. 21, presents some features of special interest.

This patient had squinted ever since she was two years of age. The affection was supposed to have supervened on her having received a fright.

The vision in each eye was equal to $\frac{2}{20}$ letters.

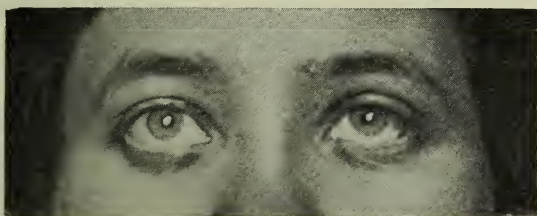
The amount of squint measured by projection and sexagesimal (not metre) tangents was nearly 20° . The right eye alone was used for fixation, although at an earlier age the affection seems to have been alternating. Examination of the fields of vision showed that of the right to be in all respects normal. The test-object used was a very small letter moved round the arc of a Priestley Smith's perimeter. The right eye



Miss P. L. before operation.

was found to move both to the right and to the left in a perfectly normal manner. On the other hand there was very defective action in the rotation of the left eye towards the left, although the rotation of this eye towards the right was perfectly normal.

As was to be expected in a case in which the vision was so good in either eye, this patient had at times diplopia.



Miss P. L. after operation.

Little need be said about the photographs of this case except that in both the patient was turned full face to the camera, and that the fixation was for distance. Unfortunately the view taken before operation has not been well focussed, but it shows the squint quite plainly.

The external rectus of the left eye alone was advanced.

Fourteen days afterwards the stitches were removed. The patient has now no diplopia, and she has binocular fixation to about four inches from her nose.

The next case, Mrs. M., is even more striking.

This patient was operated on in another town for convergent strabismus some years ago. The operation selected was teno-



Mrs. M. before advancement.

tomy of the left internal rectus, and it was twice performed on the same eye. Now, the surgeon who operated on this patient is a man of very high reputation as a practical surgeon. It would be the height of nonsense to suppose that an operation performed by him would not be thoroughly and efficiently done. Yet the final result was that the patient was left with a most disfiguring amount of convergent strabismus.



Mrs. M. after advancement.

No blame whatever can be attributed to the surgeon in this case by any fair-minded person; at any rate not at the time at which the operation was performed, for it was long prior to the advocacy of advancements at all.

Looking at the photograph of Mrs. M. before advancement it is obvious that the axes of the two eyes meet at a very

short distance in front of the patient. Yet at the time at which the exposure was made the fixation was for a great distance (much more than 20 feet). The patient was placed somewhat obliquely relatively to the camera, but still the condition can be realized by careful examination of the picture. The convergence remaining after the performance of two tenotomies was, as measured by the perimeter, about 35° .

In this case the advancement of one external rectus muscle, the left, sufficed to bring about the improvement shown in the second photograph. When this photograph is critically examined, it will be seen how symmetrical the corneal reflex is on each eye, the fixation being for a great distance. Only one muscle was advanced. As a rule I advance both external recti for convergent squint, and that at the same time, but in this case a careful examination of the field of fixation before operation showed the functions of the internal recti to be seriously impaired. A somewhat large clinical experience has led to the conclusion that as a rule a satisfactory result is only obtained when both external recti are advanced. Only once in a great number of cases has this resulted in divergence, but in that case this slight defect was easily overcome by advancement of the internal rectus which had previously been divided, and an excellent result was ultimately obtained.

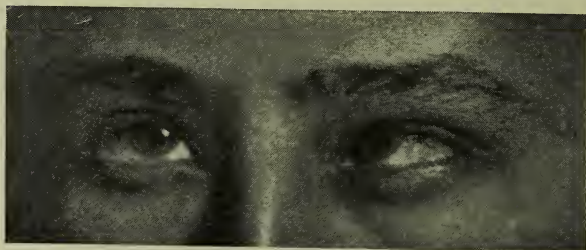
The visual acuteness of the right eye was $\frac{6}{6}$, with a hypermetropia of 3.5 D; the left eye was very amblyopic and the hypermetropia amounted to 4 D as estimated by the ophthalmoscope.

The next case is one which well illustrates the advantages of double advancement, although, for reasons which I cannot now recall, the two eyes were operated on at an interval of a week. Speaking generally, however, it may be said that the double operation should almost invariably be selected.

N. L., a lad aged 16 years, had a strabismus convergens of a most marked type. Judged by the ordinary methods of measurement it exceeded 40° . Notwithstanding this extreme amount the vision in both eyes was nearly normal, viz. $\frac{2}{30}$ in each.

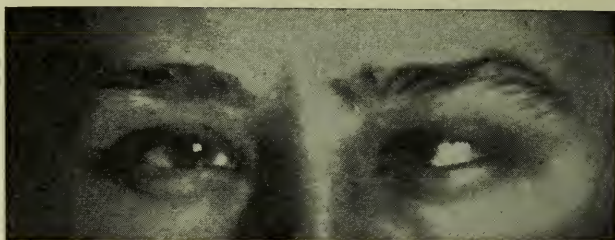
The diagrams require little explanation.

The first of the series represents the patient before he



N. L. No. 1

was operated on at all. The second was taken after the advancement of the left external rectus, and shows little



N. L. No. 2.

or no improvement, and the third is the final result after the right external rectus had also been advanced.

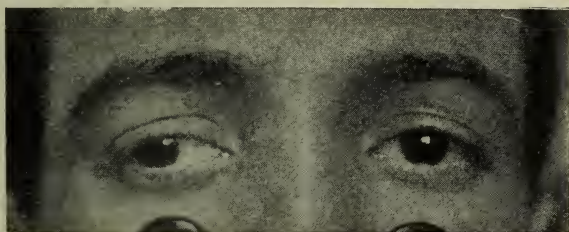


N. L. No. 3.

It has already been pointed out that, in many cases, tenotomy still leaves a marked convergence. It is but right to state that advancement occasionally does the same thing. It does so, however, much less frequently than tenotomy,

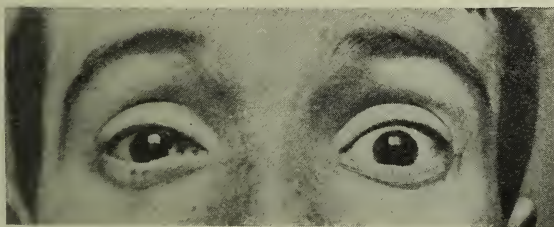
and this disappointing condition almost never occurs if both external recti are simultaneously advanced. Many more equally striking photographs could be shown of cases operated on for convergent strabismus, but these would be almost without exception a mere repetition of those already shown.

Turning now to cases of ordinary strabismus divergens, I find that the results are equally good. I do not know in the whole range of ophthalmic practice a more satisfactory operation than advancement for divergent squint, unless it be in certain cases the same operation for the relief of asthenopia due to heterophoria.



J. R. T. before operation.

Two cases are selected for demonstration chiefly because they are the first that came to hand, and illustrate all the important points.

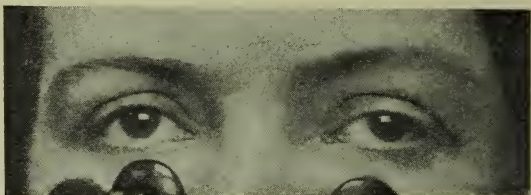


J. R. T. after operation.

J. R. T., aged 16. Patient suffered from divergent squint, the amount of deviation being between 15° and 20° . Vision in the right eye without any correction was rather less than $\frac{20}{200}$. On examination it was found that there were about 8 D of myopia in this eye, and subsequently to operation he was ordered a concave spherical glass of that strength.

The left eye had, without correction, vision = $\frac{2}{4}0$. When fitted with a weak concave cylinder the vision was $\frac{2}{2}0$ easily.

One operation only was performed, viz., advancement of the internal rectus muscle of the right eye. This was done on the 10th of April, 1899, and the patient was dismissed on the 1st of May following. A final note was made that the eyes were perfectly straight, and that the near point of binocular fixation was four inches from the nose. The test employed for binocular fixation was the simple one of covering the eyes alternately and watching for any movement of readjustment when a near point was fixed.



M. M'L. before operation.

Only one word of explanation requires to be made as to the photographs. In both the fixation was for distance. The altered appearance in the second is due to the fact that the patient was required to raise his eyebrows as much as possible in order to get the corneal reflex on the eye which had been operated on.



M. M'L. after operation.

M. M'L., aged 28. This patient had a divergent squint which was said to have been present since early childhood. The angle of squint when admitted to the hospital was found to be about 15° .

The vision in the right eye was $\frac{2}{5}$ without any correction of errors of refraction, and that of the left was $\frac{2}{7}$.

An examination of the fields of fixation showed that there was distinct limitation in the movement of the left eye towards the right side. The patient was dismissed twelve days after advancement of the left internal rectus, with the eyes quite straight, and with a near point of binocular fixation at seven inches from her face—about six metric angles of convergence.

These cases are only samples of a large number which might have been given. In the Glasgow Eye Infirmary alone I have in the course of the last four years performed the operation of advancement no fewer than 130 times. In no case has there been any trouble in the operation itself, except in one in which the stitches gave way on the day following the operation. Even in that case, however, ultimately a good result was obtained by subsequent operation. No eye has been lost, and there have been no cases of tenonitis. I have seen suppurative tenonitis involving the loss of the entire eye in a case operated on by tenotomy by a surgeon who denied the septic origin of post-operative inflammation, and whose methods of procedure were regulated accordingly.

Silk sutures are in the main to be preferred to catgut. If properly prepared by immersion in 1-20 solution of carbolic acid for a prolonged period, they can be kept in the conjunctiva for an indefinite length of time without causing any inflammation. The explanation possibly is that the epithelium grows into the substance of the thread, completely preventing any septic material finding its way along it into the tissues.

In conclusion, it may be said that before any operation is undertaken or indeed even contemplated, the patient should be thoroughly examined as to refraction, fields of fixation, and positive and negative ranges of convergence. Without a thorough inquiry into the nature of the case, any operation must largely partake of the nature of a haphazard experiment.

PERTUSSAL GLUCOSURIA, WITH OBSERVATIONS
ON THE REDUCTION OF FEHLING'S SOLUTION
BY THE URINE IN CHILDHOOD.

By R. S. THOMSON, M.D., B.Sc.

THE observations which I purpose to record in the present communication were commenced fourteen years ago, their primary object being to investigate the existence as well as the characters of that condition occasionally referred to in medical literature under the name of "Pertussal Glucosuria."

From this main inquiry, however, I have been compelled to extend my observations over a much wider field, so as to bring within their scope the conditions presented by the urine in a large number of diseases other than pertussis with the object of throwing light on the true character of the so-called "glucosuria" of whooping-cough.

The attention of the medical profession was first directed to the reducing power of the urine in whooping-cough upon copper salts by Dr. Gibb, of London, in a paper on the "Pathology of Saccharine Assimilation," published in the *Lancet* in 1855. In this paper Dr. Gibb gives no details, but contents himself with stating that sugar "is occasionally present in the urine of whooping-cough, both in its simple and complicated forms." In a letter to the *Lancet* of 1858, Dr. Gibb confirms his previous observation, and refers to the remarkable effect of nitric acid in bringing about the disappearance of the sugar from the urine. In the same publication (Vol. I., 1858, p. 120) there is a further reference to "Pertussal Glucosuria" among the "Clinical Records." A

corroboration of Dr. Gibb's observation is also published at p. 302 of this volume by Dr. Johnston, of Birmingham, who, however, while admitting the presence of sugar in the urine of whooping-cough patients, does not consider it peculiar to the disease, much less does he think it the cause of the affection. In his opinion sugar is present in the second and third stages of nearly every case of whooping-cough, while it is absent in the first. Like Dr. Gibb, this observer gives no details of observations. With the exception of a few general references to the subject scattered through the text-books devoted to diseases of children, I have been unable to discover any further references either in general terms or in detail. Dr. Pepper, of Philadelphia, has likewise failed to discover any literature on this subject beyond what has been quoted here.

The practical absence of detailed observations upon a subject which appears to have created a considerable amount of interest when first enunciated by Gibb and Johnston is probably due to the fact that whooping-cough, while it is excluded from general hospitals on account of its infectious nature, is not commonly admitted to such hospitals as are devoted exclusively to the reception of zymotic diseases, on account of its generally accepted trifling character and prolonged course. As a result, opportunity for long continued and detailed observation is wanting. In the City of Glasgow Fever Hospital at Belvidere, however, considerable accommodation is provided for the isolation and treatment of whooping-cough cases, and here a large amount of material is available. In the wards of this institution my observations were commenced, but as collateral points and side issues had to be followed up, and experiments on doubtful points extended and corrected, I was compelled to draw my material from other sources, and the Western Infirmary, the Royal Hospital for Sick Children, the Kennedy Street Fever Hospital, and the Small-pox Hospital were all laid under contribution.

At first sight it would appear a comparatively simple matter to place upon record a series of observations demon-

strating the existence of "pertussal glucosuria," but when we consider the difficulties which beset the usual tests for glucose, and more especially Fehling's test; when we have to decide whether the reducing action on Fehling's solution of the urine of those suffering from whooping-cough is peculiar to that disease, or is possessed by the urine from other diseases, as well as from healthy children, and when we have to decide the nature of the substance present in the urine which confers upon it the power of reducing Fehling's solution, the extent of the subject will be better appreciated.

From what has been said it will be obvious that the observations about to be recorded aim at the solution of the following questions:

I. Has the urine of persons suffering from whooping-cough a reducing action upon Fehling's solution, and if so, is this reducing action a frequent one, and what are its relations to the phases of the disease?

II. Is this reducing action of the urine in whooping-cough peculiar to that disease, or does urine from children suffering from other diseases possess a similar reducing power?

III. In the event of this reducing action being possessed by the urine of children suffering from diseases other than whooping-cough, is this reducing action *greater* in whooping-cough than in those other diseases?

IV. Is this reducing power possessed by the urine of healthy children?

Before proceeding to discuss my observations in detail it will conduce to a clearer appreciation of the subject if I give a description of the method of carrying out Fehling's test which was almost uniformly followed, and which reduces to a minimum the risk of fallacy. Fehling's solution to the amount of 6 c.c. is placed in a test-tube $6'' \times \frac{5}{8}''$, and plunged into hot water until the temperature reaches 205° F., when 4 c.c. of the suspected urine are added, and the temperature again raised to the same point. If urine so treated contain 0.25 per cent. of sugar immediate reduction takes place, the contents of the tube becoming opaque and

slightly yellow, while if it contain less than this quantity of sugar—and if the contents of the tube be allowed to cool *very* gradually, after a variable period depending on the amount of sugar present, and sometimes extending to several hours—the contents become milky and opaque, but *not* yellow. If a tube with such contents be allowed to stand, however, for a period of say twenty-four hours, a deposit of *hydrated suboxide of copper*, forming a yellow spot varying in size according to the extent of the reduction, will be found at the bottom of the test-tube, while the upper part of the fluid continues opaque and turbid. The substance in the supernatant fluid deposits very slowly and may continue suspended for many days. This may be mistaken for earthy phosphates, but that it is the result of the presence of glucose may be readily demonstrated by previous fermentation when the power of producing this reaction is lost by the urine, so treated, if sugar be present in small amount only. This method of determining the presence of suboxide of copper only after the lapse of twenty-four hours was the result of experience, as I found that a much larger proportion of the urines examined after that period gave evidence of reduction than when the contents of the test-tubes were examined on complete cooling—say at the end of one or two hours. It might be objected that this method is open to fallacy, and that changes might be produced in the test solution by substances other than sugar present in the urine, as a result of prolonged contact; but experiments on this point convince me that this objection is entirely unfounded. The importance of allowing the mixed fluids to stand for so long a period as twenty-four hours will be evident if we take into consideration the high specific gravity, 1.162, of Fehling's solution, which even when mixed with a proper proportion of urine reaches 1.104, and the extremely fine state of subdivision in which the precipitated suboxide exists.

The milky appearance presented by Fehling's solution on the addition of a minute quantity of glucose-containing urine is quite characteristic, though it can be distinguished from earthy phosphates only after long experience in working with the

test ; but the deposition of hydrated suboxide of copper after some hours will enable those who have had any ordinary experience with Fehling's solution to at once detect even the slightest reduction. The importance of this method may be readily demonstrated by shaking up the contents of a test-tube, showing a slight reduction, when it will be found that the suboxide re-collects at the bottom of the tube very slowly, occasionally only after an interval of several hours. The milky appearance above referred to is not invariably present even when the reducing agent is glucose, for sometimes the supernatant fluid is free from suspended particles and quite transparent, the suboxide forming a well-defined patch at the bottom of the test-tube. When a urine contains a mere trace of glucose, reduction occurs very slowly, even in the presence of a great excess of the test-fluid. This fact also makes it a matter of importance not to decide upon the presence or absence of sugar till after the lapse of some hours. When the reducing action of a urine depends upon the presence of a minute quantity of glucose the resulting suboxide is, so far as my experiments go, invariably yellow and never red. This point is in itself one of some importance, as the precipitate which results from the reduction of Fehling's solution by other substances is often unlike this, varying in colour from a brick-red to purple, or even vermilion, the last colour occasionally being produced by prolonged heating of even freshly prepared test-fluid. The following table, which gives the mean result of a number of experiments intended to bring out the gradual precipitation as well as colour of the suboxide, may prove of interest. The first series of experiments was carried out in aqueous solutions of glucose of varying degrees of strength, while in the second series of experiments normal urine was substituted for the water. In every case 3 c.c. of solution of glucose were treated with 4 c.c. of Fehling's solution.

It will be seen from this table that glucose in its interaction with Fehling's solution behaves differently in some respects according as it is dissolved in water or in urine. The reaction appears practically the same except in point of colour until

Percentage of Glucose.	Solution in Water.	Percentage of Glucose.	Solution in Urine.
1	Immediate, brick-red ppt.	1	Immediate, yellow ppt.
0.5	Immediate, brick-red ppt.	0.5	Immediate, yellow ppt.
0.25	Immediate, brick-red ppt.	0.25	Immediate, yellow ppt.
0.125	Brick-red ppt. after 1 min.	0.125	Yellow ppt. after 1 min.
0.0625	Brick-red ppt. after 3 min. Supernatant fluid almost clear.	0.0625	Yellow ppt. after 3 min.
0.031	Purplish deposit at bottom of test tube in 5 min. Supernatant fluid slightly turbid.	0.031	Yellow ppt. on becoming quite cold.
0.016	Very slight deposit when the contents of the test-tube were quite cold.	0.016	Yellow ppt. on becoming quite cold.
0.008	Minute trace of deposit when the urine was quite cold.	0.008	Slight yellow ppt. after 12 hours.
0.004	Very slight ppt. after 6 hours.	0.004	No reaction after 24 hours.
0.002	No reaction after 24 hours.	0.002	No reaction after 24 hours.
0.001	No reaction after 24 hours.	0.001	No reaction after 24 hours.

the solutions are reduced to a strength of 0.031 per cent. when we find the aqueous solution giving a more marked difference to the colour of the resulting suboxide, viz., a purplish rather than red tint, which is maintained through all the subsequent degrees of dilution, while the precipitate deposited by the urinary solution of glucose is, without exception, bright yellow. In the second place, while aqueous solutions of glucose precipitate the suboxide with great rapidity, in most instances leaving a clear supernatant fluid, the urinary solutions when they contain a comparatively small quantity of glucose deposit the suboxide much more slowly, leaving, it may be for many hours or even for several days, a milky and very opaque supernatant cloud, which deposits very slowly. These facts furnish a rough method of estimating approximately the quantity of glucose present in a sample of urine where the former does not exceed 0.25 per cent., because any urine which contains less than that amount shows no signs of reduction until it has been allowed to stand for some little time, and they indicate a ready method by which

we may gauge the intensity of reducing power possessed by a sample of urine. In future I shall speak of those reductions which take place while the test is being performed as "immediate" reductions, while those which take place after some little time and which indicate less than 0.25 per cent. of glucose as "remote" reductions.

Though most of the experiments were carried out as just described, it was occasionally inconvenient to employ a water bath for heating the tubes, and under these circumstances the usual method of heating over a spirit lamp or Bunsen flame was employed, care being taken that the contents of the tube were elevated to the boiling point and immediately removed from contact with the flame.

All urines were examined for evidence of reduction immediately on the test being carried out as well as at the end of twenty-four hours.

In deciding the nature of the substance or substances present in the urine which conferred upon it the power of reducing Fehling's solution with the separation and precipitation of cuprous oxide, the fermentation test had to be employed very extensively.

This test, when used for the detection of minute quantities of sugar, requires to be carried out with the greatest possible care on account of certain fallacies with which it is beset. In the first place, urine when passed even without coming into contact with the air contains something like 15 volumes per cent. of gas which may be readily collected by the mercurial pump, or less completely by merely heating the urine. This amount is increased when the urine is passed in the usual manner and allowed to stand in contact with the atmosphere. This gas is still further increased in amount when the urine contains even a minute quantity of sugar, as a result of the spontaneous fermentation which the urine undergoes under such circumstances. In the second place, the yeast, whatever variety be used, almost invariably contains a small amount of gas in its interstices, as well as traces of sugar clinging to its substance. To prevent fallacy arising from gas derived from these various sources, this gas and sugar must be com-

pletely got rid of before we are able to draw any definite conclusions from the results of the fermentation test.

The plan usually followed by myself consists in placing the urine to be examined in a small flask capable of holding from 50 to 100 c.c., provided with a long, narrow neck having a capacity of from 3 to 4 c.c. To this a tube provided at the upper end with a thistle funnel of considerable capacity is fitted by means of a perforated rubber stopper, so that the lower end reaches to the bottom of the flask. This flask is filled to the lip with urine to be examined, and the stopper inserted in such a way that all air is excluded and the urine lies in accurate contact with the stopper. The displaced urine rises into the funnel. The whole is then plunged into a pan of water and boiled until gas, which after a few minutes collects in considerable quantity, ceases to be evolved. The flask and contents are then allowed to cool, when the gas is permitted to escape by loosening the stopper.

The apparatus when cool is ready to receive the yeast. This latter is prepared by breaking up either dry or compressed German yeast into small particles, placing them in a beaker with distilled water and exposing for twenty-four hours in a water-oven at a temperature of from 30° to 35° C. As a result of this proceeding, any sugar clinging to the substance of the yeast is destroyed, and all gas in its interstices is got rid of. The yeast, after being thus treated, is washed in repeated changes of tepid water till the washings cease to give an acid reaction to litmus paper. Yeast so prepared may be kept for several days under water, and is entirely pure and free from objection as a test-medium, as may be demonstrated by placing a little in freshly boiled distilled water and exposing in a water-oven for twenty-four hours. On boiling it no gas will be evolved.

A little of this yeast, the amount depending upon the quantity of urine to be fermented, is then placed in the flask prepared as above, and the whole exposed in a water-oven and maintained at a steady temperature of 30° to 35° C. for twenty-four hours, though in some cases thirty-six hours may be allowed. Care, however, must be taken that the

fermentative process is not carried on for too long a time, as putrefactive changes will occur in the urine and in the yeast, and gas will be evolved as a consequence. After the lapse of at least twenty-four hours the flask, if no accumulation of gas is apparent in the neck, must be again placed in boiling water and kept at the boiling point for half an hour or longer, when the gas in solution will be driven off and collect in the narrow neck, part of the fermented urine being displaced into the bulb of the thistle funnel. The amount of gas should be noted after complete cooling of the apparatus, and may be proved to be carbon dioxide by testing with lime water. If the urine should contain no sugar, and if the test be carried out as directed, absolutely no gas will be given off. In conducting the fermentation test for the detection of minute quantities of sugar, boiling after fermentation is absolutely essential in view of the fact that at a temperature of 60° F. urine is capable of dissolving about its own volume of gas. In mixing the prepared yeast with the urine, agitation of the contents of the flask must be carefully avoided. If the neck of the flask be graduated the amount of gas may be used as a somewhat rough method of estimating the quantity of sugar.

The fermentation test carried out as described is one of considerable delicacy, and, as will be seen from the following table, is capable of detecting very minute quantities of sugar either in water or in urine:

Percentage.	Solution of Glucose in Water.	Solution of Glucose in Urine.
1	Free evolution of CO ₂ after 24 hours.	Free evolution of CO ₂ after 24 hours.
0.5	Slight evolution of CO ₂ after 24 hours.	Slight evolution of CO ₂ after 24 hours.
0.25	No CO ₂ after 24 hours, but evolved freely on boiling.	No CO ₂ after 24 hours, but evolved freely on boiling.
0.125	CO ₂ evolved on boiling.	CO ₂ evolved on boiling.
0.063	Small amount of CO ₂ evolved on boiling.	Small amount of CO ₂ evolved on boiling.
0.032	Small amount of CO ₂ evolved on boiling.	Small amount of CO ₂ evolved on boiling.
0.016	No reaction and no evolution of gas on boiling.	No reaction and no evolution of gas on boiling.

The results of actual experiments upon weighed quantities of anhydrous glucose given in this table bear out the theoretical results when the solubility of carbonic dioxide in water is remembered, that solubility making it impossible that gas should be evolved at ordinary temperature with quantities of glucose less than 0.5 per cent. The value of heating the fermented fluid and the delicacy of the test when properly applied will be seen from the table. The fermentation test may be rendered more delicate for the detection of minute quantities of sugar by concentrating the suspected urine and filtering before submitting it to the fermentative process. Concentration seems to interfere little or not at all with the activity of the yeast.

Another method of applying the fermentation test consists in evaporating the urine to a small bulk by boiling, then filtering while hot. This gets rid of any coagulable substance, such as albumen, which may be present. The filtered urine is then evaporated to dryness on a water bath, and the residue treated with boiling alcohol, filtered and again evaporated to complete dryness. The final residue is then treated with warm water and fermented as already described.

When performing the yeast test it is always well to prove the activity of the yeast used by fermenting with it a solution of glucose in water.

The destructive action of yeast upon glucose by fermentation may be taken advantage of to determine the presence of *small* quantities of that substance in urine. After twenty-four hours' fermentation in a water-oven urine so treated fails to reduce Fehling's solution even after prolonged standing. When the quantity of glucose is large this method is not applicable, as it is almost impossible under these conditions to get rid of the last trace of sugar.

THE REDUCING POWER OF THE URINE IN WHOOPING-COUGH.

Having considered the available literature on "Pertussal Glucosuria," and the chemical methods adopted in these

investigations, I shall now proceed to explain the routine followed in collecting the samples of urine, confining myself for the present to the urine of the whooping-cough patients. The reducing power of the urine in the disease under consideration was investigated in eighty-two cases, of which thirty-eight were male and forty-four female patients. The ages ranged from seven months to ten years. In twenty of the cases every sample passed by the patients under observation was examined, and occasionally as many as eleven samples from a single patient were tested in the course of the day. In the remaining sixty-two cases two, or sometimes three samples only were examined—one at 8 a.m., while the patient was fasting, and a second at 8 p.m., representing the urine during digestion. When a third sample was collected it was taken about noon. My object in examining every sample in the first set of cases was to determine if possible the relation of the reducing power of the urine to the “whoop” and cough; but the amount of labour this involved to the nurses compelled me to abandon it after the completion of the first twenty cases.

The period of residence of the patients in hospital extended from three weeks to three months, so that the number of samples examined in different cases varied considerably, the greatest number tested in any one patient being 385. In the course of my remarks I shall use the words “night” and “day” to denote the periods embraced between 8 p.m. and 8 a.m., and between 8 a.m. and 8 p.m. respectively.

In all, 7376 samples of urine were examined, or an average of 90 from each patient. Of these, 5185, or a little over 70 per cent., gave no evidence of *immediate* reducing power on testing, while 2191, or about 30 per cent., gave very decided and *immediate* evidence of reducing power during the process of testing.

I would especially emphasize this point, because a careful distinction must be drawn between the reducing power of the urine as manifested while testing,—it is to such reductions I have applied the term *immediate*, and the same power as manifested by the precipitation of suboxide of copper *after*

cooling or standing for several hours. These I have termed *remote reductions*.

Of the total samples, 3642, or about 50 per cent., deposited suboxide on standing for twenty-four hours, or during the process of testing. Of these reductions two-fifths were *remote*.

The difference of time occupied in the separation and precipitation of the suboxide means a difference in amount or intensity of reducing power, which must be borne in mind when comparing the extent of the reducing power of the urine in whooping-cough with that found in the urine of diseases other than whooping-cough, because this difference shows a variation in the quantity of the substance or substances to which the urine owes its power of reduction.

While the *immediate* reducing power of the urine in about 30 per cent. of the patients examined was very decided and often extremely well marked, the appearance of the precipitate sometimes resembling that got with true diabetic urine, yet in few of the samples subjected to volumetric analysis did I find more than a reducing power such as would be represented by a 1 per cent. solution of glucose, *i.e.* about $4\frac{1}{2}$ grains per ounce. In the great majority of samples it was much less than this, and in many cases an accurate estimate of the reducing power was scarcely possible. In no case was the specific gravity markedly affected, and in *none* did the quantity of urine suggest diabetes.

The proportion of urines possessing *immediate* reducing power to the total samples examined in any individual case varied within wide limits; in some of the cases almost every sample gave a reaction with the test solution, while in others the proportion fell as low as 9 per cent. Not only did the reducing power vary in individuals, but it also varied in the same individual from day to day.

Before proceeding to analyse the various stages of whooping-cough in their relation to the reducing power of the urine, it will be best to discuss the more general features of the subject, taking eighteen out of the total cases as samples.

This will enable me to deal more readily with the statistical side of the question, as I shall in this way avoid an unwieldy

mass of figures which would prove cumbersome and difficult to deal with, and would obscure the subject rather than render it clearer. These eighteen cases have been selected from the others on account of their freedom from complication, and will be used to illustrate the subject throughout.

The following table gives a general view of the number of samples examined in these cases with the proportion of those showing *immediate* reducing power, as compared with those in which no such power was manifested.

The relation of the reducing power of the urine to the severity of the attack is shown in the last column.

Case No.	Total Samples.	Reducing.	Not Reducing.	Proportion of Reductions.	Type of Case.
1	95	55	40	58 %	Severe.
2	110	22	88	20 %	Mild.
3	34	14	20	41 %	Severe.
4	83	27	56	32·5 %	Moderate.
5	234	158	76	67·5 %	Very severe.
6	155	67	88	43 %	Severe.
7	383	131	252	34 %	Moderate.
8	229	111	118	48·5 %	Severe.
9	228	80	148	35 %	Mild.
10	245	94	151	38·4 %	Moderate.
11	203	38	165	18·5 %	Mild.
12	220	75	145	34 %	Moderate.
13	142	13	129	9·7 %	Very mild.
14	328	176	152	53·7 %	Very severe.
15	97	20	77	20·5 %	Mild.
16	132	76	56	57·5 %	Severe.
17	58	32	26	55 %	Severe.
18	62	22	40	35·5 %	Mild.

An examination of this table will show :

(1) That a large proportion of the urines examined gave an *immediate* reduction.

(2) That the reducing power varies enormously in the different cases, the proportions ranging from 67·5 per cent. down to 9·7 per cent.

(3) That the proportion of urines manifesting *immediate* reducing power is directly as the severity of the attack.

In an investigation such as the present the question whether the dietary of the patient may not be to some

extent responsible for the reducing power of the urine naturally suggests itself.

The dietary in all the cases was very similar, consisting of milk and farinaceous food with a comparatively small amount of meat. While it is obvious that the dietary will influence the whole of the urine passed in each period of twenty-four hours, yet we would expect to find its influence upon the urine more marked during the day than during the night. This point must be borne in mind when we come to discuss the influence of the diet upon the reducing power of the urine in whooping-cough, as compared with other diseases. In the following table an attempt is made to show the influence of night and day upon the reducing power of the urine. In the

Case No.	DAY.			NIGHT.			PROPORTION PER CENT.	
	No Reduction.	Reduction.	Totals.	No Reduction.	Reduction.	Totals.	Day.	Night.
1	24	30	54	16	25	41	56	61
2	41	12	53	47	10	57	22·6	18
3	8	8	16	12	6	18	50	33·3
4	32	15	47	24	12	36	32	33·3
5	38	80	118	38	78	116	67·5	67
6	53	30	83	35	37	72	36	51
7	128	78	206	124	53	177	38	30
8	68	45	113	50	66	116	40	59
9	80	29	109	68	51	119	27	43
10	104	44	148	47	50	97	30	51
11	106	17	123	59	21	80	14	26
12	107	32	139	38	43	81	23	53
13	86	5	91	43	8	51	5·5	15
14	106	78	184	46	98	144	42	68
15	39	10	49	38	10	48	20	20
16	25	41	66	31	35	66	62	53
17	12	18	30	15	14	29	60	49
18	21	8	29	19	14	33	27	42

first section (day) the urines examined were those of children in whom the digestive processes and absorption were being carried on actively, while in the second section (night) urines from fasting children only are dealt with. The first column of each section shows the number of urines in which no reduction was obtained, and the second the number of urines in which reducing power was manifest. The third column gives

the total urines passed in each period of twelve hours under observation. The proportions of reductions during the day and night are compared in the last column of the table.

On comparing the figures in the last double column it is evident that though the proportions between "day" and "night" reductions are quite inconstant, yet the figures show a reducing power decidedly in favour of the fasting urines, and on working out the mean of these proportions it is found that the reductions during day and night are as 37 to 42, the preponderance being in favour of the fasting urines. This fact, so far as it goes, is decidedly against the suggestion that the reducing power of the urine is the outcome merely of dietetic conditions, for, as already hinted, if it were so we would expect the reductions to be more frequent and intense while the processes of absorption and assimilation are most active. The difference, however, is too slight to form a basis for anything like a definite conclusion.

No. of Case.	ABUNDANT.		CONSIDERABLE.		DISTINCT.		TRACE.		MIN. TRACE.	
	Day.	Night.	Day.	Night.	Day.	Night.	Day.	Night.	Day.	Night.
1	0	0	5·5	0	11	20	18·5	25	11	9
2	0	0	0	0	13·6	13·6	18	18	13·6	23
3	0	0	0	0	7	14·5	14·5	43	21·5	0
4	0	0	0	0	25·9	37·1	14·8	19	3·7	0
5	0	0	0	0	29	28	16·5	17·7	3·8	5
6	0	0	0	0	15	17	26	21	8	13
7	0	0	0	0	12·5	28	17	23·5	8·5	10·5
8	0	0	0	0	20·5	24	16·5	25	3·6	10·4
9	0	0	0	0	2·5	10	15	36	18·5	18
10	0	0	4·3	1·1	20	13	18	23	5·5	17
11	0	0	0	0	8	18·5	18·5	26	18·5	10
12	0	0	0	2·6	17	30·5	20	17	5	8
13	0	0	15	0	0	30	15	15	10	15
14	0	0	0	2	14	23	20	22	10	9
15	0	0	0	0	5	0	20	30	25	20
16	0	0	6·5	6·5	14	9	18	29	6·5	9
17	0	0	0	0	6·5	19·5	19·5	42	9·5	3
18	0	0	0	0	0	4·5	27	45	9	14·5

It may be suggested that although the urine from fasting whooping-cough patients shows a more *frequent* reducing power than that from the same patients while digestion is active, yet that this is merely the "tailing off" of the more

active reduction present during the day as shown by a *greater* instead of a more frequent precipitation of suboxide. The preceding table bears on this point, and is intended to show the relation between the urine passed during the day and night periods, and the reducing power as indicated by the quantity of suboxide precipitated. In this table reductions both "immediate" and "remote" are dealt with. The proportions are in percentages, and refer to the number of samples examined.

A glance at this table will make it clear that, so far as it can be gauged by the amount of suboxide precipitated, there is no constant relation between the *intensity* of the reducing power in the urines passed during periods of fasting and those passed during active digestion. But while this is so, on examining the mean of the separate columns we find that the figures in each stand as follows :

	Day.	Night.
"Considerable,"	7·8	3·1
"Distinct,"	14·2	20
"Trace,"	18·5	26·5
"Minute Trace,"	10·6	12

Here we have it demonstrated that the reducing power of the fasting urine is decidedly greater than that of the urine passed during active digestion, except in the case of the column headed "Considerable," in which, however, the number of reductions is too small to warrant our drawing any definite conclusions.

If we exclude the "Considerables" and compare the mean of the "day" column of "Trace" with that of the "night" column of "Distinct," and the mean of the "day" column of "Minute Trace" with that of the "night" column of "Trace" (see preceding table), we find the figures stand thus :

Night.		Day.	
Distinct,	20	Trace,	18·5
Trace,	26·5	Min. Trace,	10·6

This comparison of the reducing power of the fasting or "night" urine with that of the succeeding period of active digestion also shows in favour of the greater *intensity* of the reducing power of the former. It thus becomes evident that the fasting urines of whooping-cough possess not only a more *frequent*, but also a more *intense* reducing power than

those passed during active digestion. This may be the result of the greater concentration of the urine passed during the night than during the day.

Among the eighty-two cases of whooping-cough investigated there was one in which there was no indication of a "whoop" throughout the whole course of the attack, so that it became impossible to separate the stages of the illness. This case was an extremely mild one. From it 157 samples of urine were examined, of which 63 were fasting urines and 94 were collected during the day. "Immediate" reductions were given by 24, or 15·3 per cent. of the whole; and none of the reductions indicated a marked intensity of reducing power, 58 per cent. of these being recorded as showing only a trace of suboxide.

Before passing from the consideration of the general features of my subject, it may be of interest to give a brief summary of a case which came under my notice suffering from whooping-cough, and two years later with varicella. The condition of the urine of this child while suffering from the former disease was very striking when compared with that while suffering from varicella. During the course of its first illness no less than 69·5 per cent. of the samples examined gave *immediate* reduction, while during the attack of chicken-pox 52 samples of urine were examined, of which only 3, or about 6 per cent., gave *remote* reductions, and even these were indicated by the presence of only a minute trace of suboxide. The diet of this child was practically the same during both periods of its residence in hospital. This striking difference may have resulted from the difference of age at the two periods of examination.

Passing from our discussion of the more general points associated with the reducing power of the urine in whooping-cough, the influence of the various stages of the disease on this power now falls to be considered.

For obvious reasons but little opportunity was afforded for examining the urine during the first or catarrhal stage, but in the case of four of the children who came under observation when the disease was merely suspected, owing to other

members of the family suffering from fully developed and typical attacks, this opportunity was presented. In all four *immediate* reducing power was manifested, the extent of which will be best appreciated by a reference to the subjoined table, which shows the number of samples examined during the catarrhal stage with the proportion of urine possessing *immediate* reducing power.

Case.	Samples.	Reduced Fehling's Solution.	Per Cent.
1	20	4	20
2	23	3	13
3	25	9	36
4	41	23	56

From this it will be seen that during the catarrhal stage the urine does possess reducing power of sufficient intensity to produce an *immediate* precipitation of cuprous oxide in 39 of the 109 samples examined, or a mean proportion of nearly 36 per cent.

Leaving the observations on the second or "convulsive" stage for future discussion, it will be most convenient at this point to give details of the reducing power presented by the urine during the third stage, or that of "resolution." Here we have abundant material on which to base our conclusions. In one case of great severity the child was removed from hospital, and was lost to observation before the completion of the second stage of the attack, and, as already mentioned, it was impossible in one case to discriminate between the different stages of the illness on account of the absence of "whoops."

This leaves us with 80 cases in which it was possible to note the condition of the urine during the stage of convalescence. Taking into consideration only the cases in which the end of the convulsive stage was moderately well defined, we find that of the urine passed during this period, *i.e.* between the complete cessation of "whoop" and dismissal from hospital, 25 per cent. gave evidence of reducing power

capable of causing *immediate* precipitation of cuprous oxide on heating with Fehling's solution. It would serve no good purpose to give here a detailed list of the condition of the urine in every case during this stage, and in the accompanying table I shall again consider the condition present in the urine of those eighteen cases which I have already discussed in the earlier part of this section.

Case No.	Total Samples.	Total Reduction.	Proportion.	Case No.	Total Samples.	Total Reduction.	Proportion.
1	39	10	25·6 %	10	70	36	51·5 %
2	60	5	8·4 %	11	91	26	28·5 %
3	29	4	13·4 %	12	71	10	14 %
4	32	5	22·7 %	13	39	10	25·5 %
5	21	15	71 %	14	113	9	8 %
6	101	54	53·5 %	15	14	8	57 %
7	101	36	35·6 %	16	25	5	20 %
8	221	34	15·8 %	17	38	22	58·5 %
9	156	39	25 %	18	28	11	39 %

The figures here detailed afford a fair sample of all of the cases investigated. It will be observed on reference to the table that the proportion between the number of urines showing reducing power and those which did not possess this power varied within wide limits, viz., from 8 to 71 per cent., and in general it may be said that these proportions were a direct index to the severity of the attack—Case 5 in table being a very severe one, while Case 14 was of the mildest possible type, a “whoop” having been present for only some three days. Case 6 is an example of a patient detained a long time in hospital on account of the continuance of the cough, and from whom a very large number of samples was examined during the stage of “resolution”—no less than 53·5 per cent. of them showing reducing power in a very high degree.

The mean of the proportions of “reducing” to “non-reducing” urines in this stage as set down in the foregoing table is 27 per cent.

Passing now to a consideration of the condition of the urine in the second or “convulsive” stage, we find that by far the greatest reducing power was manifested by the urine during

this period. Taking the mean of 80 cases in which this period was tolerably well defined, 49 per cent. of all the samples tested exercised an *immediate* or *remote* reducing action upon Fehling's solution.

This fact is well brought out in the subjoined table, a glance at which will show that, like as in the stage of

Case No.	Total Samples.	Total Reduction.	Proportion of Reductions.	Case No.	Total Samples.	Total Reduction.	Proportion of Reductions.
1	56	45	80 %	10	152	68	44·7 %
2	50	17	34 %	11	136	28	20·6 %
3	19	13	68 %	12	181	65	36 %
4	62	12	19·5 %	13	29	4	14 %
5	133	104	78·2 %	14	339	165	48·5 %
6	54	8	14·8 %	15	27	10	37 %
7	162	97	60 %	16	49	12	24·5 %
8	73	72	98·5 %	17	70	46	65·7 %
9	163	44	27 %	18	31	21	68 %

“resolution,” the reducing power of the urine varies greatly in different patients, the highest reducing power being shown by Case 8 with 98·5 per cent. of reductions, and the lowest by Case 13 with only 14 per cent. The reducing power of the urine in this stage, however, is at once seen to be much greater than in either the first or third stage, the sequence of proportions being 46·5 per cent. in the convulsive, 36 per cent. in the catarrhal, and 27 per cent. in the resolvent stages respectively. A comparative view of the reducing power of the urine in the various stages of the disease in the 18 cases already analysed is given in the accompanying table, from which it will be seen

Case No.	Catarrhal Stage.	Convulsive Stage.	Resolvent Stage.	Case No.	Catarrhal Stage.	Convulsive Stage.	Resolvent Stage.
1	—	80	25·6	10	—	44·7	57·5
2	—	34	8·4	11	—	20·6	28·5
3	—	68	13·4	12	—	36	14
4	—	19·5	22·7	13	—	14	25·5
5	—	78·2	71	14	—	48·5	8
6	20	14·8	53·5	15	13	37	57
7	—	60	35·6	16	36	24·5	20
8	—	98·5	15·8	17	56	65·7	58·5
9	—	27	25	18	—	68	39

that, while the averages of the reducing urine in the three stages are as stated above, viz., 36 per cent. in the catarrhal, 46·5 per cent. in the convulsive, and 27 per cent. in that of resolution, yet when we examine the individual cases we do not find this order invariably maintained as we might expect if the reducing power is to be considered a characteristic feature of the convulsive stage, and due to conditions peculiar to this period of the illness, for we observe that while in twelve of the patients the urine during the convulsive stage possessed greater reducing power, in five this power was greatest during the stage of resolution, and in one during the catarrhal stage. From this it is evident that the reducing power cannot be the result of conditions present only during the convulsive stage of whooping-cough, and it follows that this reducing power is not to be considered as the result exclusively of such phenomena as are present during this stage, that is to say of the greater or less degree of venous obstruction produced by the respiratory convulsion, nor yet to any other condition associated with the "whoop," except in so far as the severity of the convulsive stage directly measures the gravity of the whole attack; and though it is difficult to demonstrate this from figures, yet there seems to be little doubt that the more severe the attack the greater is the reducing power of the urine in all its stages. With a view to the elucidation of this point, a number of careful observations were made to determine whether there existed any direct relation between the reducing power of the urine and the coughs associated with the "whoop," as distinguished from those without the "whoop." An attempt is made to show this relationship in the accompanying scheme, though it is clearly impossible to determine the actual effect of a "whoop" upon the reducing power of any given sample of urine. This scheme gives details of 8 cases in which careful notes of the coughs with and without "whoop" were made during each period of 24 hours, and its object is to demonstrate as far as possible any association between the "whoop" itself and the reducing power of the urine.

The first column (day) refers to the day of observation, not

to that of illness; and under each case the four columns give, first, the number of whoops (W.); second, the total coughs (C.); third, the number of urines showing reducing power (R.); and fourth, the total number of samples examined during the corresponding period of 24 hours.

An analysis of the figures in this table shows that the reducing power of the urine is maintained even after the complete disappearance of the "whoop," as will be apparent on comparing the period of "resolution" (as gauged by the disappearance of the "whoop") of Cases 1, 3, and 5. In all the cases noted, except 1, 4, and 8, the number of "whoops" was very great at the commencement of the period of observation, yet if we exclude Case 7 during the first six days, more than one-half of the samples of urine examined from these cases showed no reducing power. On comparing these again with Cases 1, 4, and 8, in which the "whoop" was much less frequent, we do not find that in these cases the proportion of samples manifesting reducing power was less than in the former group. Finally, in comparing the earlier periods during which the "whoop" was most frequent with the later periods, when it was much less frequent and less severe, we sometimes find the reducing power even more strongly marked in the latter than in the former period, as may be seen in Case 2, 22nd to 26th day of observation, and in Case 3, from the 18th to the 24th day.

From a consideration of these facts it would seem legitimate to conclude that neither the "whoop," apart from the other conditions manifested by whooping-cough, nor the obstruction to the venous circulation, which is generally more marked in cases where the "whoop" is most frequent, can be considered to influence the reducing power of the urine.

But though it seems clear that while the "whoop" in itself, or the convulsive cough associated with it, has no direct connection with the reducing power of the urine, it may be argued that the frequency of the coughs, both such as are associated with "whoops" and those which are not, may have some direct influence upon the reducing power.

A reference to the foregoing scheme, however, will show

that the number of coughs has no closer association with this reducing power than the "whoops" themselves have.

Again, it may be suggested that any greater reducing power shown by the "night" urine is the result of the greater frequency of the "kinks" at night as compared with the day.

The following table is a record of the coughs in nine patients who were carefully observed, and the coughs recorded by competent nurses who for years had been in charge of whooping-cough wards. A separate record was kept of the coughs during the night and day.

The figures here set down support my earlier impression, that in point of fact the symptoms of whooping-cough are not more severe nor the "kinks" more frequent at night than during the day. This opinion I formed as the result of a long series of observations carried out some years ago to ascertain the "specific" action of certain drugs in the treatment of whooping-cough. I had occasion at that time to examine this question in close upon 200 cases, and in these no constant nocturnal exacerbation could be demonstrated. The nine cases given in the table are entirely in favour of these former conclusions, and show that while sometimes the "kinks" are more frequent and severe at night, yet just as often the reverse is the case, and there can be but little doubt that in whooping-cough the "kinks" are tolerably evenly distributed between day and night. The suggestion, therefore, that a greater severity of the attacks at night will account for the greater frequency, as well as the greater intensity of the reducing power at this period, cannot be entertained.

So far we have discussed the reducing power of the urine in the different stages of whooping-cough, its relation to the whoop, and its daily variations, and now further points suggest themselves as to the relationship of certain other conditions, such as age and sex, to this reducing power. As already stated, the ages of the patients ranged from seven months to ten years, and in the following short table these have been arranged year by year up to the eighth year; the

CASE 1.		CASE 2.		CASE 3.		CASE 4.		CASE 5.		CASE 6.		CASE 7.		CASE 8.		CASE 9.	
Day.	Night.	Day.	Night.	Day.	Night.	Day.	Night.	Day.	Night.	Day.	Night.	Day.	Night.	Day.	Night.	Day.	Night.
5	3	4	2	27	33	9	14	11	10	10	14	9	4	15	17	4	7
4	7	3	1	13	19	9	12	8	14	9	11	12	4	16	11	11	8
7	2	0	0	14	16	9	12	6	11	11	5	14	5	14	10	6	2
6	3	1	1	12	13	11	13	9	10	13	2	13	2	14	8	8	3
3	1	0	0	13	12	7	12	11	9	11	0	13	5	13	4	5	4
2	3	0	0	12	10	5	12	12	8	10	5	13	5	11	5	3	0
1	1	0	0	9	11	9	11	11	8	9	2	10	4	9	8	1	2
1	1			7	11	4	9	8	4	10	1	6	4	6	3	2	3
2	2			14	14	5	2	6	5	6	2	5	3	9	3	2	0
2	0			6	8	5	5	16	8	5	0	6	3	7	2	0	0
2	0			8	7	8	8	11	6	6	0	9	1	9	5	1	
0	0			8	4	3	6	8	5	8	1	7	2	4	2		
				9	8	7	8	12	8	3	1	4	2	1	2		
				3	7	5	7	13	5	1	0	2	2	2	5		
				12	7	4	8	11	5	0	0	0	1	3	3		
				6	9	1	10	12	7	1	0	0	3	5	2		
				5	6	7	4	8	5	0	0	0	0	3	5		
				3	7	4	4	8	7	5	0	6	3	3	2		
				6	7	3	1	14	3	1	0	4	4	5	3		
				4	8	3	0	10	5	4	2	6	2	6	9		
				8	5	1	1	11	5	5	3	6	1	8	5		
				4	6	0	4	9	4	0	1	4	3	5	5		
				6	6	1	0	11	6	0	3	8	6	6	6		
				3	6	1	0	11	6	0	1	3	0	7	0		
				3	6	1	0	6	2	0	0	6	0	0	0		
				8	4	0	0	4	2	0	0	1	3	0	3		
				2	8	0	0	6	4	0	0	0	1	0	0		
								6				0	3		0		

last entry includes the small number of cases from eight to ten years :

Age Period.	Proportion of <i>Immediate</i> Reductions to Total Samples.					
0-1 year,	-	-	-	-	-	59·5 %
1-2 years,	-	-	-	-	-	No cases
2-3 „	-	-	-	-	-	33 %
3-4 „	-	-	-	-	-	40 %
4-5 „	-	-	-	-	-	53 %
5-6 „	-	-	-	-	-	35·5 %
6-7 „	-	-	-	-	-	35 %
7-8 „	-	-	-	-	-	32 %
8-10 „	-	-	-	-	-	16 %

The proportion of reductions to the total samples of urine examined at the different ages given in this table would not suggest the existence of any relation between the reducing power of the urine and the different age periods ; but I would call attention to the first age period with a proportion of 59·5 per cent. of *immediate* reductions. The cases represented in this age period were all *sucklings*, and, as will be seen later, the urine of such patients usually possesses a high reducing power, so that it is difficult in the present instance to decide how much of that power is traceable to the dietary, and how much to the whooping-cough.

While there is no steady fall in the proportion of reductions as age advances, yet there is, if anything, a tendency in this direction. It is important here to remember that these proportions represent *immediate* reductions only.

Sex would seem to have quite as little influence as age on the reducing power of the urine.

As has been already stated there were thirty-eight males and forty-four females, and an examination into this point reveals nothing noteworthy, the mean proportion of urines showing *immediate* reducing power being practically the same in both sexes.

We have discussed the reducing power in general, its variations in the different stages of the disease, the relations of this power to the cough and "whoop," as well as its variations at different periods of the day, and lastly its relations to age and sex, and consequently we are now in a position to answer in

a fairly satisfactory manner the first of the questions with which we set out, viz.: "Has the urine of persons suffering from whooping-cough a reducing action upon Fehling's solution, and if so, is this reduction a constant one, and what are its relations to the phases of the disease?" The evidence brought forward so far warrants the following conclusions:

1st. The urine of children suffering from whooping-cough, both in its simple and in its complicated forms, possesses a decided reducing action upon Fehling's solution. This power is not possessed by every sample of urine, although 30 per cent. of all samples examined manifested a power of causing an *immediate* reduction of the copper solution; or in other words, that proportion of samples possesses a power of reducing Fehling's solution such as would be represented by a solution of glucose containing 0.25 per cent. and upwards, while in addition about 20 per cent. of the samples possess a reducing power less than this.

2nd. This reducing power varies in its intensity and in its frequency of occurrence, but is most markedly developed in the severe cases, being apparently in direct proportion to the gravity of the attack. The reducing action of the urine was at some time manifest in all the patients examined.

3rd. This reducing power is not present to the same degree in all phases of the illness, being most marked during the convulsive stage, and less so in the first and last stages.

4th. The "whoops," except in so far as they are an index of the severity of the attack, do not appear to influence the reducing power of the urine either in regard to its intensity or its frequency of occurrence.

5th. Diet would seem, unless in *sucklings*, to exercise a doubtful influence upon the reducing power, for fasting urine shows considerably greater activity in this respect than that passed while digestion is active.

6th. The reducing power of the urine being doubtfully influenced by diet, and having no apparent relation to cough "whoop," or other variable features of the disease, may be regarded as *possibly* connected with the pathological conditions which produce the diseased state.

This brings us to our second and third questions, viz.: "Is the reducing action of the urine in whooping-cough, which we have been discussing, peculiar to this disease, or does the urine from children suffering from other diseases possess a similar reducing power; and in the event of this reducing action being possessed by the urine of children suffering from diseases other than whooping-cough, is this reducing action *greater* in whooping-cough than in those other diseases?"

These questions can be answered only by an analysis of the urine in a variety of diseases peculiar to childhood; and in this connection a series of observations on the urine in the zymotic diseases and others more or less prevalent in the earlier years of life will now be detailed. Of these, the first to be dealt with is varicella. This will be followed in detail by scarlet fever and measles, in all of which diseases, owing to their segregation in hospital wards, it was possible to carry out an extensive series of observations. In other diseases, such as those of the heart, lungs, intestinal canal, nervous system, etc., where isolated cases only were available for observation, the same opportunity of examining masses of cases was not afforded; so that while the records of these cases have been made as extensive as possible, they lack that completeness and force of evidence possessed by the first class of cases.

THE REDUCING ACTION OF THE URINE ON COPPER SALTS IN CHILDREN SUFFERING FROM CHICKEN-POX.

The reducing action of the urine of chicken-pox patients upon Fehling's solution was investigated in 99 cases. In each case, with a few exceptions, two samples were examined—passed about 8 a.m. and 8 p.m. respectively. The period of residence of the patients in hospital varied from 21 to 70 days, so that the number of samples likewise varied; the smallest number investigated was 17 and the greatest 188 in any one case—while the average for the whole was a little over 70 samples. Occasionally there was great difficulty experienced

in collecting samples at regular intervals from the younger patients, and in these, sometimes only one daily sample was available.

These 99 cases represent a total of 7134 samples examined, of which 4632, or about 65 per cent., gave no evidence of reduction; whilst 2502, or 35 per cent., gave distinct evidence of reducing power in varying degrees. In only two of the cases examined there was no evidence of reducing power manifested by the urine; while in the individual cases the number of samples showing reducing power varied from 1, representing 2·3 per cent. of the samples examined, up to a maximum of 101—representing 99·8 per cent. of the total samples examined. Though this latter percentage (99·8) is high, I would here call especial attention to the important fact that only a very small percentage of all samples (3·9) manifested this reducing power while the test solution and urine were being heated, in this respect showing a marked contrast to whooping-cough, in which 30 per cent. of the samples examined showed this *immediate* reducing action. Even this small percentage of *immediate* reductions would have been much smaller but that in three cases the urine possessed such marked reducing power that the *immediate* reductions represented 1·2 per cent. of the total *immediate* reductions occurring among the samples examined. Of the balance of the reductions (31·1 per cent.) the reducing activity of the urines was represented in most cases by a mere trace of precipitated suboxide, and that only after the test tube had been allowed to stand for some hours, *i.e.* these were all *remote* reductions. The reducing power varied greatly, not only in the different individuals, but also in the same individual from time to time.

An interesting phenomenon was observed in some cases, namely, that the reducing power of the urine was manifested at irregular intervals in the course of the cases, the samples possessing reducing power being in the majority of instances not scattered uniformly through the period of observation, but grouped together, a single case showing one or more groups, each group preceded and succeeded by a period of variable length during which the urine showed no trace

of reducing power. Another point noted was that these groups of urines manifesting reducing power did not coincide with the more acute phase of the illness, but frequently occurred towards the end of the attack, or even after convalescence had been completely established.

In the case of those children who were being nursed by their mothers the reducing power of the urine was very strongly marked, practically no day passing while the children were under observation without one, and often both samples examined showing very marked reducing power. Bearing in mind that all the cases of chicken-pox observed lived under practically the same conditions and partook of practically the same kinds of food, except of course in the cases of the sucklings, the marked variation of the reducing power in the different children would suggest the question whether some idiosyncrasy of nutrition might not be responsible for the high reducing power possessed by the urine of some of the patients.

Going a little more into the details of the cases we find that when these are arranged in age periods we have the following results :

Age Period.	Reductions Per Cent.					
0-1 year	-	-	-	-	-	58
1-2 years	-	-	-	-	-	39
2-3 „	-	-	-	-	-	39
3-4 „	-	-	-	-	-	33
4-7 „	-	-	-	-	-	28
7-13 „	-	-	-	-	-	22

I am unwilling to insist too much upon these figures, yet they are of interest, and strongly suggest that for some reason the urine of very young children possesses a much higher degree of reducing power than that of older children. As already stated, the food and surroundings of all the cases observed were practically the same, except in the cases of sucklings, so that the conditions of reduction with reference to age suggested by this table can hardly be attributed to difference of food, and this circumstance would lead us to infer the probability of some gradual alteration either in the process of digestion and absorption or in the more obscure and less

familiar metabolic processes connected with the liver or general tissues.

Sex would seem to make little difference, so far as my statistics show, in the reducing power of the urine in chicken-pox; but, so far as they go, the figures are in favour of a higher reducing power among males than females. Among individual males the percentage of reductions varied between 0 and 99·8 per cent., and among females between 0 and 68 per cent.; while a mean of all the observations gave 37 per cent. for males and 33 per cent. for females—*i.e.* a percentage of 4 in favour of the former. The sexes taken separately and examined in age periods reveal nothing noteworthy.

As already explained in an earlier part of this paper, the urine collected at 8 a.m. represented the absorption and metabolism in the fasting child, while that collected at 8 p.m. represented the absorption and metabolism of the child while the processes of digestion and absorption were active; and we might hence expect that these morning and evening urines would show some decided difference in reducing power. In some individual cases there was indeed a marked difference, but this was neither constant in different individuals nor in the same individual from day to day, and in some there was exactly the same average during each period of twelve hours. Taking the mean of all samples examined the difference practically disappears, the percentage of evening reductions, representing the process of digestion, being 51, while 49 represents that of the morning or fasting urines.

THE REDUCING ACTION OF THE URINE ON COPPER SOLUTIONS IN CHILDREN WITH SCARLET FEVER.

The reducing action of the urine in scarlet fever was investigated in 52 patients, but as five of these cases were incomplete, the remarks which follow will apply to the remainder, 47 in number. The samples were collected at eight in the morning and at the same hour in the evening, so that the latter represents the urine of the period of digestion. In

all, 1609 samples were examined, divided in nearly equal proportions between the two periods mentioned. Of this number, 1082 gave no evidence of reducing power, while in 527 the evidence of reducing power was unequivocal. This gives a proportion of 30·37 per cent. of reductions. On analysing the number of urines which manifested reducing power, we find that of the total only 15, or 3·4 per cent., gave an *immediate* reaction, the other reductions being *remote*. In other words, of the whole number of samples examined only 1·2 per cent. gave *immediate* reductions—in this, as in the case of chicken-pox, contrasting strongly with whooping-cough, in which the *immediate* reduction formed 30 per cent. of the total samples examined. These *immediate* reductions occurred in eight of the cases, leaving 38 in which every reduction was *remote*. In one case no single sample gave evidence of reducing power, and in a second case every sample reduced the test solution with facility. Thus the frequency of the reducing power ranged from 0–100 per cent., and between these extremes every degree of reducing power was represented. So far as could be judged, the stage of the illness had absolutely no influence on the frequency or extent of the reductions. The *degree* of the reducing power seemed to vary greatly in different individuals, as was also the case with the *frequency* of reductions, both being best seen among the younger children, though here also individual peculiarity seemed to play an important part, for in certain of the children the reductions were constantly much more marked than in others.

The period of the day would seem, in the case of scarlet fever, to exercise quite a decided influence upon the reducing power of the urine, and this influence was such as to produce a markedly higher reducing power among the “night” than among the “day” samples. Of the total samples, 799 were collected in the morning and 810 at night, the former representing the fasting samples, and the latter those passed while digestion was active. Of the former, 280, or 35 per cent., manifested reducing power, while of the latter, 247, or 30·5 per cent., possessed the power of

throwing down suboxide. These figures show that the reducing power of the "night" or fasting urines was about 5 per cent. greater than that of the urine passed during the day. The children examined included 23 males and 24 females, and the samples tested were derived, 797 from the former and 812 from the latter. Of the samples collected from the males, 264, or 35.6 per cent., possessed distinct reducing power, while of those collected from females, 263, or 32.3 per cent., reduced the test solution. The relation of the reducing power to age among children suffering from scarlet fever emphasizes the points brought out by the examination of this question in the case of varicella, as will be seen from the accompanying table, in which the ages are arranged in periods of one year. The youngest child whose urine was examined was two years of age and the oldest twelve.

Age.					Reductions Per Cent.
0-1 year,	-	-	-	-	No cases.
1-2 years,	-	-	-	-	No cases.
2-3 "	-	-	-	-	69.7
3-4 "	-	-	-	-	53.6
4-5 "	-	-	-	-	47.0
5-6 "	-	-	-	-	39.0
6-7 "	-	-	-	-	42.0
7-8 "	-	-	-	-	23.0
8-9 "	-	-	-	-	18.0
9-10 "	-	-	-	-	20.9
10-12 "	-	-	-	-	7.7

There were no sucklings among the cases observed in this disease, but the gradual fall in the reducing power of the urine with increasing age is very striking. This feature, it will be recollected, was not strikingly marked in the cases of whooping-cough.

THE REDUCING POWER OF THE URINE ON COPPER SOLUTIONS IN CHILDREN WITH MEASLES.

A detailed analysis of all the diseases examined, on the lines followed in varicella and scarlet fever, would occupy too much space and extend this paper beyond reasonable limits.

I will therefore summarize my results and give as concisely as possible the conclusions to which I have come, contrasting in tabular form the number of cases examined in each disease, and the reductions both *immediate* and *remote* produced by the samples.

The urine was examined in 93 cases of measles, but owing to the fact that the experiments were carried out early in the course of my investigations, in a considerable number a record was kept of the *immediate* reductions only. In those examined later, both *immediate* and *remote* reductions were noted. The samples were collected at the same hours as in the diseases already recorded, and the circumstances, surroundings, and food of the patients were practically the same in every instance.

The *immediate* reductions which, as already stated, were recorded in all the cases amounted to 1.45 per cent. of the samples examined, the number of which exceeded 5000, while the *remote* reductions, which, however, were recorded in only a small proportion of the cases, amounted to no less than 57.5 per cent. This latter proportion is possibly too high, and further observations are desirable. As in the diseases already discussed, the "night" or fasting urine possessed greater reducing power than the "day" urines, the proportions being 67 per cent. reductions among the "night," as compared with 53 per cent. among the "day" urines. With regard to sex and age, the same points were noticeable as in the other diseases. Sex apparently had little influence on the reducing power, while the same marked reducing action among the younger children, as compared with the older, was evident, as in chicken-pox and scarlet fever.

REDUCING POWER OF THE URINE ON COPPER SOLUTIONS IN CHILDREN WITH DIPHTHERIA.

The urine was examined in five cases of this disease—two males and three females—with a total of 168 samples, equally divided between the periods of day and night. Of these, 105

gave no evidence of reducing power, while 63 reduced Fehling's solution more or less actively. This gives the proportion of 37·5 per cent. of urines showing reducing power. Of these reductions 8 were *immediate*, equal to 4·7 per cent. Individual peculiarity apparently was responsible for many of these reductions, seeing that 53 out of the total 63 reductions occurred in two of the patients, the balance being divided between the other three. This individual peculiarity is strikingly brought out by the fact that all the *immediate* reductions, 8 in number, occurred in the same individual. Of the two cases referred to above, one was a male, the other a female; in the former 90 per cent. of the samples, and in the latter 72 per cent., showed reducing power. The night and day reductions were equal in number, being 37·5 per cent. of the total samples during each period of 12 hours.

We have now completed our analysis of the observations carried out upon the urine of children suffering from zymotic diseases, and it now remains to discuss the reducing power of the urine in children suffering from non-infectious diseases. As already remarked, the investigations into this class were less complete and continuous than in the infectious diseases owing to the absence of segregation. It would be quite impossible in this place to give complete details of all the cases examined, and I shall therefore content myself with stating in a tabular form those points only which bear most directly upon the facts brought out in the section on whooping-cough, this being, after all, the main object of our inquiry. The annexed table gives all the necessary details, and is arranged, the first part, in the order of frequency of the *immediate* reductions, while the latter part, containing those diseases in which the urine showed no *immediate* reducing power, is arranged in the order of frequency of the *remote* reductions.

A study of this table reveals several points of great interest. Confining our attention in the meantime to the figures indicating the proportion of *immediate* reductions, we see that whooping-cough, which is essentially a convulsive disease, heads the list with 30 per cent., and is followed, though at

Table showing the Reducing Power of the Urine in different Diseases, with "Immediate" and "Remote" Reactions.

Name of Disease.	No. of Cases.	Immed. Reactions.	Rem. Reactions.	Total Reactions.	Remarks.
Whooping Cough, -	82	30 %	20 %	50 %	
Gen. Convulsions, -	3	14·3 %	0 %	14·3 %	
Epilepsy, -	5	10 %	0 %	10 %	
Pleurisy, -	15	5·2 %	13·8 %	19 %	
Enteric Fever, -	17	5 %	40 %	45 %	
Dis. of Spinal Cord, -	13	5 %	28 %	33 %	
Tub. Peritonitis, -	9	4·8 %	33·2 %	38 %	
Psoriasis, -	10	4·4 %	31·1 %	35·5 %	
Diphtheria, -	5	4·7 %	32·8 %	37·5 %	
Anaemia, -	151	3·9 %	25·3 %	29·2 %	
Gastric Catarrh, -	63	3·2 %	27·1 %	30·3 %	
Tonsillitis, -	15	3 %	20 %	23 %	
Varicella, -	99	3·9 %	31·1 %	35 %	
Bronchial Catarrh, -	134	2·3 %	33·7 %	36 %	
Tub. of Bone and Glands, -	95	2·2 %	21·8 %	24 %	
Small-pox, -	3	2·2 %	43·8 %	46 %	{ One case red. 100 %.
Intestinal Catarrh, -	126	2 %	28·9 %	30·9 %	
Bronchitis, -	100	2 %	28 %	30 %	
Rickets, -	82	1·6 %	26·4 %	28 %	
Measles, -	93	1·45 %	57·5 %	59 %	
Scarlet Fever, -	47	1·2 %	29·17 %	30·37 %	
Chronic Abscess, -	39	1·2 %	28·8 %	30 %	
Healthy Children, -	364	1 %	23·5 %	24·5 %	
Eczema, -	149	0·7 %	35·7 %	36·4 %	{ One case gave red. 60 %.
Phthisis, -	34	0·5 %	38·1 %	38·6 %	
Chorea, -	36	0·5 %	29·5 %	30 %	
Burns, -	2	0 %	100 %	100 %	{ One case red. 80 %.
Cardiac disease, -	16	0 %	62·2 %	63·2 %	
Scabies, -	2	0 %	55 %	55 %	
Pseudo-Hyper. Par., -	1	0 %	54 %	54 %	
Pemphigus, -	5	0 %	53 %	53 %	
Cirrhos. of Liver, -	1	0 %	50 %	50 %	
Pneumonia (Croupous), -	4	0 %	50 %	50 %	
Tabes Mesenterica, -	6	0 %	50 %	50 %	
Cerebral Tumour, -	1	0 %	40 %	40 %	
Laryngitis, -	3	0 %	33 %	33 %	
Syphilis, -	2	0 %	33 %	33 %	
Mumps, -	10	0 %	30 %	30 %	
Neuralgia, -	7	0 %	29·2 %	29·2 %	
Tubercular Meningitis, -	2	0 %	27·2 %	27·2 %	
Strophulus, -	2	0 %	25 %	25 %	
Nephritis, -	10	0 %	24·4 %	24·4 %	
Herpes, -	3	0 %	24 %	24 %	
Rheumatism, -	15	0 %	10 %	10 %	
Broncho-Pneumonia, -	10	0 %	5·3 %	5·3 %	

a considerable distance, by two other conditions, both characterized by convulsive seizures, viz., general convulsions (no doubt resulting from various causes) and epilepsy. In these diseases the intensity of the reducing power is very marked, though in both, convulsions and epilepsy, the total *frequency* is not great. Chronic diseases of the nervous system unassociated with convulsions likewise show a tolerably high intensity of reducing power as well as a very considerable frequency.

Those diseases in which irritation of the skin and mucous and serous membranes is a prominent feature, possess a marked intensity, and in some cases, as in diphtheria and tubercular peritonitis, a high frequency of reducing power.

Another point which is worthy of attention, especially in view of the suggested explanation that the reducing power of the urine in whooping-cough, is due to the cerebral congestion caused by the cough itself, is the fact that the urine in diseases associated with cough such as bronchitis, bronchial catarrh, and croupous pneumonia, does not manifest a reducing power upon Fehling's solution above the average, and in broncho-pneumonia shows a *remote* reducing power of only 5.3 per cent.

It would be difficult to say how much of the reducing power of the urine was due to the diseases from which the children suffered, but the fact that the great majority of the diseases investigated showed reducing power in about 30 per cent. of the samples (5) would suggest that this percentage approximately indicates the average of reducing power independent of diseased conditions; but I would again call attention to the important part played by idiosyncrasy. This last point has been already referred to in several instances, as will be seen by turning to the preceding pages.

The table furnishes an answer to the question whether the urine of presumably healthy children possesses a distinct reducing power on Fehling's solution. Altogether the urine was examined in 364 children, all of whom failed on careful examination to give any indication of general disease. Children with naevi, slight ring-worm, and other slight *local*

affections were regarded as healthy for the purpose of this investigation. In the cases examined exactly 1 per cent. of the urines gave *immediate* and 24·5 per cent. *remote* reductions. We are therefore in a position to assert with confidence that not only does the urine in diseased conditions possess reducing power, but that the urine of healthy children likewise manifests such power in a very marked degree. It would thus seem probable that in the urine of children there is present in variable amount, but more or less constantly, some substance (glucose) which possesses the power of precipitating cuprous oxide from alkaline solutions of copper salts.

CONCLUSIONS.

An analysis of the facts brought out experimentally in the course of the observations which have been recorded points in the direction of the following conclusions:

1. A large proportion of the urines in children suffering from whooping-cough possess the power of reducing Fehling's solution.

2. This reducing power is present in all the phases of the disease, but is more marked, both with regard to its intensity and frequency, in the spasmodic than in the other stages.

3. This power of reducing alkaline solutions of copper salts is not peculiar to whooping-cough, and is possessed in varying degree by most other diseases of childhood; but it is much greater in intensity in whooping-cough than in any other of the diseases investigated. The urine in every case of whooping-cough examined possessed at times a high reducing power, though the frequency with which this power showed itself varied within very wide limits.

4. The urine of presumably healthy children also possesses the power of reducing copper salts, but the frequency of reduction is less than in whooping-cough.

5. The most important point of difference between the reducing power of the urine in whooping-cough and that derived from children suffering from diseases other than

whooping-cough, and from healthy children, lies in the greater intensity of the reducing power of the urine in the first of these, seeing that 30 per cent. of whooping-cough urines manifested reducing power such as would be possessed by a 0.25 per cent. solution of glucose or upwards.

6. The reducing power of the urine in whooping-cough is manifested with greater certainty, and with less reference to age, than either in health or in diseases other than whooping-cough.

7. From the fact that the urine of whooping-cough manifests reducing power in all stages of the diseases, and in attacks not associated with the "whoop," also from the fact that the whoop has little influence except in so far as it measures the severity of the affection, the question arises, whether the reducing power of the urine is due to the pathological changes which give rise to the various symptoms constituting the disease, seeing it is not directly caused by the passive congestion of the nerve centres, incident more especially to the spasmodic stage. This conclusion is strengthened by the observation that the urine in diseases associated with marked cough, such as bronchitis, does not present a reducing power strikingly beyond the average.

8. The reducing power possessed by the urine of whooping-cough is probably invariably due to the presence of glucose.

A CASE OF CYSTIC TRANSFORMATION OF THE KIDNEYS AND LIVER.

By CHARLES WORKMAN, M.D.,

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GLASGOW Royal Infirmary Pathological Reports, No. 2205. Date, 3rd March, 1899. The patient, James T., aged 34, a lorryman, was admitted to the wards on 27th February, 1899, as an "urgent abdominal case."

He was quite evidently very ill, and complained of abdominal pain. He had been ill for at least three months, but had got very much worse in the ten days previous to admission. He had had no vomiting, no diarrhoea, and no headache. On admission he got frequent enemata with good effect, and they gave great relief. The abdomen on admission was greatly distended, and hard masses were felt on both sides, more especially, however, on the right. It was thought that the liver was much enlarged. The urine was small in amount and contained blood and abundant albumen, but no casts. Patient progressed fairly well till the evening of 1st March, when he became drowsy, then showed twitchings, and later became blind. The twitchings increased and ended in convulsions and death about 4.5 a.m. on 2nd March, in spite of active treatment. The symptoms were typically uraemic. (Report from Ward.)

Post-mortem Inspection.—External Appearances.—A well-developed and well-nourished body. Post-mortem rigidity pronounced. Pupils dilated and equal.

Thorax.—The pericardium contained about 1½ oz. of clear

serum. The heart was considerably enlarged, especially from dilatation and hypertrophy of the left ventricle, and weighed 15 oz.

The aortic and pulmonary curtains were competent, and these valves presented healthy appearances. The mitral orifice had a circumference of 115 mm., the tricuspid of 160 mm., and these valves also appeared healthy. The muscular tissue of the heart's wall was of good consistence and colour, and the coronary arteries, though a little atheromatous, were not narrowed. The right ventricle was slightly dilated.

The left lung was fairly voluminous and healthy; it was free from pleural adhesions. The right lung was adherent from old pleurisy, but otherwise presented healthy characters.

Abdomen.—The stomach was large and considerably distended. The mucous membrane of its cardiac end was softened by post-mortem digestion. The duodenum appeared healthy. The pancreas was large and healthy. The liver was somewhat enlarged (68 oz.) and rather pale; on careful examination it was found to present translucent areas due to the presence of cysts, generally about the size of small peas, which were scattered singly, and appeared to number only a dozen or so in the whole organ. The gall-bladder contained fluid bile, which escaped readily into the duodenum on pressure. The spleen was normal in size, $4\frac{1}{2}$ oz., but rather pale.

The kidneys had undergone a most extreme cystic transformation, being enormously enlarged; the right weighed 67 oz., the left 65 oz. They appeared to be made up of an immense number of translucent bladders, varying in size from that of a pea to that of a small orange. The contents of these cysts varied greatly in colour and consistence; some were almost black, others dark red, some straw-coloured, and others like milk or cream; some were quite fluid, others of the consistence of jelly, while some had the appearance of pus, and others the consistence of caseous material in various stages.

This gave to the organs a most remarkable appearance, resembling masses of conglomerate made up of pebbles of very various colours. The cysts containing red and black



material appeared to have extravasated blood in varying quantity and condition in them.

Microscopic sections were prepared from several parts of the kidney, and these show that the cysts are mostly lined with a single layer of squamous epithelium; and that between the cysts there is still a considerable amount of comparatively healthy kidney substance with tubules and Malpighian tufts. Sections have also been made of the cysts in the liver, and these show a structure almost identical with those in the kidneys.

The most generally received theory as to the origin of this form of transformation is that the cysts are retention cysts, caused by obstruction in the urinary tubules, giving rise to distension either of the Malpighian capsules or of portions of the tubules.

In the case which I have described this explanation is untenable, for the condition has affected not only the kidneys, but also the liver. Further, on carefully examining microscopic sections of the liver, I find that the structure of the cysts with their lining epithelium is entirely different from that of the bile ducts, and also from that of the liver acini; the very small cysts are readily seen to have no connection with the neighbouring ducts, and to be lined with a single layer of squamous or tessellated epithelium, while the ducts are very distinctly lined with columnar cells.

Microscopic examination of sections from various parts of the kidney, shows that, though at some places the cysts appear to have taken origin in the tubules, they as often seem to have no connection with either the tubules or the Malpighian capsules. The tubules, which are present in great numbers in the tissue between the cysts, are very irregular in size and shape, and their epithelium is often in various stages of degeneration. Apparently the irritation of the cyst formation has caused in many places considerable inflammatory reaction, evidenced by masses of leucocytes and the presence of much new-formed connective tissue. The Malpighian capsules and tufts, though frequently much deformed by pressure, are present in large numbers.

Probably the condition is very slow in its progress, and in this case it seems to have given rise to no suspicion of renal disease till almost the end. From this fact, along with the histological structure of the kidneys and the involvement of the liver, I am inclined to consider that the condition is allied to tumour formation, being perhaps a species of adenoma. The parenchymatous and interstitial nephritis, and the distension of tubules caused by them, I consider secondary and simply an effect of the irritation and pressure of the cystic adenoma. The sections of the liver do not show any cirrhotic condition, and, indeed, except for slight hyperaemia noticeable in the sections and the presence of the cysts, the liver appeared remarkably healthy.

The presence of cysts in the liver or other organs does not seem to have been noticed by a number of those who have examined cases of cystic transformation of the kidneys, and in many it may have been absent. Pye Smyth considered that the association was merely a coincidence. Lionel Beale injected the ducts of the liver from a case of Bristowe's, and found no connection between the ducts and the cysts.

The latest accounts that I find of this condition are by von Mutach, "Beitrag zur Genese der congenitalen Cystennieren," *Virchow's Archives*, Vol. 142, page 46; and P. Schultz, *Ueber congenitale Cystennieren*, Inaug. Dissert., Halle, 1896. Both these writers appear to be considering the condition in childhood, and they both adhere to the view that the cysts are retention cysts.

A case occurring in a woman, aged 40 years, is described by Charles Kennedy, M.D., in the *Edinburgh Laboratory Reports*, Vol. III., 1891, and the etiology of the conditions is discussed. In this case also the liver was affected, and the author states that the "weight of evidence and authority is in favour of the multiple cysts of the kidneys being retention cysts"; the author evidently adheres to this view, and considers the cysts in the liver to be retention cysts of the bile ducts. Kennedy gives a list of the literature up to the date of his paper. A good account of the foreign papers on the subject will be found in Ziegler's *Beiträge zur pathol. Anat. und allg.*



Pathol., Vol. XII., by Nauwerck & Hufschmid, "Ueber das multiloculäre Adenokystom der Niere."

Along with the account of my case I give two plates, No. 1 showing the left kidney about one-third of the actual size. This is a collotype from a negative which I made from the kidney as it appears preserved by the formol method in glycerine and acetate of potash. The 2nd plate is taken with a stereoscopic camera from the same kidney, and when looked at with a stereoscope gives the natural rounded appearance of the preparation.

The left kidney and half of the right are preserved in the Museum of the Glasgow Royal Infirmary, and the other half of the right was presented to the Museum of the Western Infirmary.

RECENT VIEWS ON THE NATURE OF IMMUNITY.

By ROBERT MUIR, M.D., F.R.C.P.E.,

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So far as advance in medical science in the last decade of the century is concerned, the outstanding discoveries are undoubtedly those of the properties of the sera of highly immunized animals and of patients suffering from infective diseases—discoveries which have led to the establishment of sero-therapeutics and sero-diagnosis respectively. The demonstration of a definite action possessed by a serum against a toxine or a living organism in the conditions mentioned has altered former conceptions regarding the nature of immunity, and has opened up a new and wide field of research. The result has been the recognition of various properties—anti-toxic, anti-bacterial, lysogenic, agglutinative, etc., and the establishment of many interesting and important facts with regard to each. There has, however, been much dispute with regard to the exact nature of these properties and of their relation to one another, and the whole subject has become very complex. The only statement which applies to them all is that they are developed in the living body in the presence of bacteria or of separated toxines. They are all apparently the result of a reactive process; they are “antagonistic substances” in the widest sense—*Antikörper*. This statement in itself, however, suggests the question whether one fundamental process may not underlie the formation of all such anti-substances. The establishment of some general principle with regard to them is not only an important gain

from the scientific point of view, but is of much practical importance as pointing out the lines along which further advance may be more quickly and surely gained. An attempt to formulate and establish a general law has recently been made by Ehrlich in his so-called side-chain (*Seitenkette*) theory of immunity. Whether the theory will be found to be true in its entirety or not, it undoubtedly marks an epoch in the subject, and has already been the means of leading to fresh discoveries. It accordingly seems worthy to be expounded and considered.

Properties of anti-sera.—Without entering into details regarding the properties of serum in the conditions referred to, we may mention, for the sake of clearness, the chief facts. On the one hand, as is now well known, the serum of an animal treated with repeated doses of certain toxines acquires an antagonizing or neutralizing action against the particular toxine, *i.e.* is anti-toxic. Similar properties have been observed in the case of patients who have recovered from certain diseases, notably diphtheria. On the other hand, in certain conditions the serum acquires properties which are not exerted against the toxine directly, but against the bacteria; we may say, properties which induce changes in the organized bacterial structure—anti-bacterial properties. In order to the acquisition by the serum of properties of this second class the bacteria, in the living or dead condition, must themselves be introduced into the system, this being of course always the case to a greater or less extent in the natural disease. It is sufficient merely to mention with regard to these properties that the action may be *lysogenic*, leading to the dissolution and ultimate disappearance of the bacteria; or it may be *agglutinative*, leading to clumping of the bacteria; whilst, along with these, the *preventive* or *protective* property is closely associated. It is beyond our present purpose to discuss the relations of these in detail. What is of importance is that substances appear in the serum which have a definite action on the corresponding bacterium. To put these facts, with regard to anti-sera, in the most general manner, we have (*a*) in the presence of a toxine the production of a body which

neutralizes its action, and (b) in the presence of the bacteria the production of substances which act on bacteria of the same species. In both cases, as we shall see later, there is evidence of direct combination, *i.e.* of anti-toxine with toxine, of anti-bacterial substances with bacterial bodies. It is to be noted that in the case of all these antagonistic substances the only test we have of their existence is their physiological action. None have been obtained in a pure condition, and, though probably all are proteids of complicated constitution, even this is not known with certainty.

Relation of anti-toxine to toxine.—In considering the mode of production of antagonistic substances it is most convenient to take first the case of anti-toxines. But before doing so it will be well to consider briefly the relation of anti-toxine to toxine; in other words, how the anti-toxine acts. The two chief possibilities are (a) that the anti-toxine acts directly on the toxine, *i.e.* chemically, and (b) that the action of the anti-toxine is through the medium of the living cells, *i.e.* physiologically. It would be interesting to discuss the facts bearing on these two theories, but this has already been recently done.¹ In our opinion all the arguments go to show that anti-toxine forms a loose chemical compound with the toxine; in fact, the evidence in favour of this view seems now to be practically conclusive. On such a theory alone do the various facts with regard to the relations of the two substances receive an adequate explanation. The use of the term "chemical action" has, however, given rise to misunderstanding. This does not mean that the toxine is destroyed or broken up by the anti-toxine, merely that a chemical union has taken place. The only evidence of disappearance of the toxine is disappearance of toxic action. Now a molecule of toxine has apparently an unsatisfied atom-group by which it enters into combination with the living cells, *i.e.* has a specific affinity for part of the cell protoplasm. When toxine is combined with anti-toxine this affinity is already satisfied, and hence towards the tissues the former is rendered indifferent

¹ *Vide* Cobbett, *Journ. of Path.*, vol. vi., 1899, p. 193; also, *Manual of Bacteriology*, Muir & Ritchie, 2nd ed., 1899, p. 475.

and therefore harmless—the toxic action disappears. In the treatment of a case of diphtheria, for example, the anti-toxine introduced unites, according to this view, with free toxine in the blood, and thus prevents its combining with and injuring the tissues. Certain observations brought forward by Calmette to controvert the view advanced by Fraser regarding the chemical relation of anti-venene to snake venom, and often quoted in this connection, are not really opposed to the chemical view of the relationship. In the case of snake poison the anti-toxine is less stable than the toxine, and is destroyed at a lower temperature. Now, Calmette showed that when a certain amount of toxine had been neutralized by the addition of anti-toxine, the toxic property was restored on heating the mixture at 68°C .—a temperature sufficient to destroy the anti-toxine. The objection has been recently brought forward against this result that the time element in these experiments was disregarded, a certain period being necessary before the combination is complete, and that at the time of applying heat a quantity both of free toxine and anti-toxine was present. Martin and Cherry found, on the other hand, that when sufficient time for complete combination was allowed, no toxic action reappeared on heating. But even if Calmette's interpretation is correct, the result stated does not disprove chemical union; it only proves that the toxine is not destroyed in the *physical* sense. If two complicated compounds of unequal stability are in loose chemical union, it is quite intelligible that the less stable may be destroyed (*e.g.* by heat) whilst the more stable escapes. We repeat then, that on the chemical view advocated by Ehrlich all that is implied is that an unsatisfied atom-group in the toxine becomes satisfied on uniting with the anti-toxine. As Ehrlich puts it, the anti-toxine behaves to toxine as a lock to a key. All the facts with regard to the action of anti-toxine—more rapid in strong than in weak solutions and at higher temperatures than at lower, more efficient when the anti-toxine is injected with the toxine than when they are injected at different parts of the body, etc.—receive a satisfactory explanation on this theory.

In the case of anti-bacterial sera also, we have evidence that the anti-substance combines with the bacteria. This has been well shown in the case of agglutinins, these being used up in definite proportions in combining with the bacterial protoplasm or sheath. A similar statement probably applies to the lysogenic property (whether this really depends on agglutinins or not)—it certainly has been completely established in the closely analogous case of lysogenesis of red corpuscles described below. In short, to state the matter briefly, it would appear that the outstanding feature in the production of all anti-sera is the formation of bodies capable of effecting chemical union with the agent used—toxine or bacterium (or rather with some part of the latter). Having thus discussed these relations of the anti-substances, we may now consider how they are produced in the living body.

Origin of anti-toxines.—Ehrlich's theory is grounded upon his views regarding the nature of toxic action. He considers that living protoplasm may be represented as consisting of a central atom-group (*Leistungskern*) and related atom-groups or "side-chains" (*Seitenketten*). These side-chains have certain unsatisfied chemical affinities by which they fix molecules and bring them into relation to the protoplasm; they thus possess an important function in the cell economy, *e.g.* in nutrition. (The analogy is taken from the "benzol-ring" of organic chemistry.) Now there is evidence that a toxine—we are speaking of those toxines to which anti-toxines can be produced—has two essential atom-groups, one by which it becomes united to the protoplasm, and another to which the toxic effects are due. By the former it becomes anchored, as it were, to the cell; by the latter it produces a disturbance in the essential structure of the protoplasm, which is the physical basis of the toxic phenomena observed clinically. When however, the toxine is introduced in doses insufficient to produce toxic symptoms—as in anti-toxine production—a similar combination takes place with the side-chains of the protoplasm. This combination being fairly durable, the side-chains are lost for the physiological purposes of the cell, and are accordingly thrown off in combination with

the toxine molecules. A restoration of the side-chains takes place in excess of those lost, so that now more toxine can be introduced with impunity. The same result will follow as before—the combination S.-T. (side-chain + toxine) will be thrown off in greater quantity. By a continuation of gradually increasing the dose of toxine, there will ultimately be an over-production of the side-chains, *i.e.* in excess of the needs of the cell, and they become free in the blood-stream. When free they have the same chemical affinity for the toxine, and thus act as anti-toxine. As Ehrlich puts it: “The anti-toxine molecules are therefore the side-chains of the cell protoplasm regenerated in excess, and therefore discharged.” The process of anti-toxine production is accordingly to be regarded as a stimulation of regeneration and over-regeneration of molecules which have been lost to the cells. Although it is stated that anti-toxine molecules are side-chains, it must be specially kept in view that they are side-chains *free in the serum*. When forming part of the protoplasm, so far from acting as anti-toxine, they really constitute the means by which the cell is rendered liable to the toxic action. On the other hand, when free in the serum they combine with the toxine, and thus prevent the latter from becoming attached to the living cells. “The same substance which, when situated in the cell, is the necessary condition of poisoning, becomes the cause of cure when it passes into the blood.” (Behring.)

Such in outline is Ehrlich's theory, and we may now consider what evidence there is in support of it, and how it harmonizes with established facts. If we first of all examine the possible sources of anti-toxine, we find that they may be said to be three in number—(a) Anti-toxine may be formed from the toxine, *i.e.* may be a “modified toxine.” (b) The anti-toxine may be the result of an increased formation of molecules normally present in the tissues. (c) The anti-toxine may be an entirely new product of living cells. It may now be considered as proved that the anti-toxine is not a modified toxine. For example, it has been shown in the case of both tetanus and diphtheria that when an animal is bled the total amount of anti-toxine in the blood may afterwards be greater

than it was immediately after the bleeding, even although no additional toxine has been introduced. This shows that the anti-toxine must be *formed* by the living cells of the body. There are other facts against this theory, which need not be detailed. Apart from the direct evidence, it is scarcely conceivable that the living body should have the power of converting the various toxins into substances which act as specific anti-toxines. It may, accordingly, be stated that anti-toxines are products of the living cells, and the third possibility mentioned—also difficult to understand on biological grounds—will be disproved if the second, as enunciated by Ehrlich, is established. In discussing the evidence in support of it, the following observations may be first of all brought forward :

(1) Ehrlich's theory offers at once an explanation of the difference between active and passive immunity, *i.e.* the immunity produced by repeated injections of toxine and that produced by injection of anti-toxine already formed. As is well known, the former is of much longer duration than the latter, a fact which is easily intelligible on the view that the cells have acquired the habit of anti-toxine formation, which is really a regenerative property. In passive immunity this property will not be brought into play at all. It would, however, be difficult to explain the difference mentioned if the anti-toxine were a modified toxine. Why, on such a supposition, should not the anti-toxine disappear from the blood as quickly in active as in passive immunity?

(2) It affords an explanation of the power which the animal body has of forming a variety of anti-toxines possessing a specific relationship to the corresponding toxins. This has always been a great difficulty in explaining the origin of anti-toxines. But if the action of a toxine depends upon a chemical affinity for certain atom-groups in the cells of the body,¹ there must be as many varieties of atom-groups as there

¹ Theoretically two different toxins might have a similar combining atom-group, and in this instance their anti-toxines would be similar. This is probably so in the case of the vegetable poisons robin and ricin (Ehrlich). But as a general rule the facts are as indicated.

are toxines, and from what has been stated above it will be evident that thus the basis for a corresponding number of anti-toxines is afforded. This power of forming anti-toxines of various kinds, which appears at first an extraordinary phenomenon, is thus to be referred back to the complicated chemical constitution of living cells. On no other theory can a rational explanation of the various specific anti-toxines be given.

(3) It should be noted that the theory implies no new process in biology; it represents merely regeneration after loss. It is in fact an application of what Weigert holds to be a general law, namely, that the bioplastic or formative processes in living tissues are always brought into play by previous loss or damage (*Schädigung*). In this connection he distinguishes a purely histo-chemical *Schädigung*, where molecules are broken down or lost without functional disturbance, from a clinical *Schädigung*, where symptoms of impaired function or of poisoning follow. The formation of anti-toxines depends upon the former; toxic action is an example of the latter. Yet the difference is rather one of extent than of kind. There is one point, however, to which further reference may be made, viz. the regeneration in excess of what has been lost, the over-regeneration implied. At first sight there may appear to be something irrational in such a supposition. But if corresponding formative processes are examined an analogy will be readily found. As Weigert points out, a similar phenomenon is probably exemplified in the case of functional hypertrophy, *e.g.* of muscle. Here also an excessive breaking down, namely, of the molecules of the contractile fibrils is the first phenomenon, and this, if continued or frequently repeated within certain limits, is not merely followed by restoration or repair, but by a repair in excess resulting in actual enlargement of the fibre. True, in this case, there is no evidence that over-regenerated molecules are discharged from the cells, yet the formative process is closely analogous to that on which Ehrlich's theory depends. So also in repair of tissues, the newly formed cells are often more numerous than those which have been lost; and, further,

this over-production or excessive proliferation becomes more marked where there is a long-continued gradual loss. As an example of this may be offered the broad zone of granulation-tissue forming the wall of a chronic sinus. In all these processes there is evidence of provision for a further and greater loss—of molecules or cells, as the case may be.

In the case of anti-toxines the amount formed strikes one as enormous. It must, however, be kept in view that the only means we have of measuring the amount is a physiological one, viz. against the action of the toxine. How much *ponderable* matter this represents we have no data for estimating. Everything, however, points to its being extremely small, just as the lethal dose of toxine is almost inconceivably minute.

If these anti-toxines are merely cell molecules which have become free in the blood stream, the question may be reasonably put—have we any evidence of the existence of such in the normal tissues? An affirmative answer has been afforded in a striking way in the case of tetanus. According to Ehrlich's theory the symptoms in this disease should be due to a combination of the toxine molecules with the side-chains of the nerve-cells. Wassermann and Takaki were the first to show that this combination actually occurs. They found that in the central nerves there are atom-groups which combine with the tetanus toxine, and therefore act as anti-toxines when tested in another animal. This was shown by bruising the spinal cord and brain of a guinea-pig so as to form an emulsion, adding a quantity of the emulsion to a certain dose of tetanus toxine and injecting into another animal. In this way they found that 1 c.c. of such emulsion could protect a mouse against ten times the lethal dose of the toxine. This, however, probably gives only a rough idea of the amount of this combining substance in the central nervous system, as the conditions after death are not the same as during life. But these results, which have been fully confirmed, demonstrate the special chemical affinity of the tetanus toxine for bodies in the central nervous system. Furthermore, emulsions of other organs tested in the same way are without effect. It may also be mentioned that a corresponding result has been

obtained by injecting the toxine in the living animal. In certain cases it has been found that after a lethal dose has been administered, free toxine can be detected in all the parts of the body except in the nervous system. In the latter situation the toxine has combined with the cells, and, as will appear from what has been said above, therefore cannot be demonstrated. Regarding the exact nature of these bodies in the nervous system practically nothing is known, but it has been found that, as regards sensitiveness to high temperatures and other physical conditions, they behave exactly as anti-toxines prepared in the usual way. It accordingly appears impossible to escape the conclusion that these atom-groups or side-chains in the normal nerve-cells and the anti-toxine molecules of tetanus are the same, *i.e.* that the latter are merely free side-chains.

In the interpretation of these results, however, there has been considerable misunderstanding on the part of various workers. The sensitiveness of the nervous system to tetanus toxine has been supposed to be a fact irreconcilable with the presence in it of anti-toxine molecules. But, according to Ehrlich's theory, the two facts are really in harmony. In the living body the molecules in question form connecting links between the toxine and the cell protoplasm; it is only when they are free in the blood stream, or, as in the above experiments, artificially brought into contact with the toxine, that they act as anti-toxines, *i.e.* as protectors of living cells. Accordingly, certain experiments of Roux and Borrel brought forward as evidence against Ehrlich's theory are really not so. They showed that the lethal dose of tetanus toxine for rabbits is much smaller when the injection is made directly into the central nervous system than in the case of subcutaneous injection. This, however, only proves that by this method combination with nerve-cells by means of their side-chains is more easily effected, and is in accordance with the theory of toxic action as above detailed. The rationale of the recently introduced method of treating tetanus by intra-cranial injection of the anti-toxine implies the same principle; the anti-toxine molecules (free side-chains) combine with any

free toxine in the nervous system and prevent its union with the side-chains of the living cells,—may probably even produce some degree of disassociation when such union has taken place.

This recent work on tetanus has accordingly yielded valuable support to Ehrlich's theory. Investigations on similar lines have also been carried out in other diseases, though the results have been much less striking. Tetanus is, however, peculiar in the definite way in which the symptoms are related to a particular tissue; in most other diseases the anti-toxines or other antagonistic substances have probably a more general origin. It was found by Wassermann in the case of typhoid, and by Pfeiffer and Marx in the case of cholera, that in the early stages of immunization the spleen, lymph-glands, and bone-marrow were especially rich in the anti-typhoid and anti-cholera substances respectively. Metchnikoff, in a recent paper, advances the view that the large hyaline leucocytes, the "macrophages," are an important source of such substances. Regarding this, however, no definite statement can yet be made. In addition to these examples in which specific combining molecules have been shown to exist in the cells of the tissues, we may also mention that traces of anti-toxines have, in certain instances, been demonstrated in the fluids of the body, *e.g.* anti-venene in the bile of the ox (Fraser), diphtheria anti-toxine in the blood serum of certain animals.

So far we have considered Ehrlich's theory as applied to the nature of anti-toxines. We have pointed out how it rests on his conception of toxic action and of the relation of anti-toxine to toxine, how it explains on biological principles the formation of anti-toxines, and how subsequently to its being brought forward it has been confirmed and supported by various recently established facts of high importance. We have now to inquire whether it can be applied to anti-bacterial sera. It is scarcely necessary to repeat that such sera have a direct action not against the toxins, only against the bacteria producing them. Nevertheless the two sera have many points in common. In addition to their being produced by an analo-

gous process, viz. the repeated injection of gradually increasing doses, the laws of passive immunity apply to both—definite substances appear in the serum of the animal actively immunized, and thus the injection of such serum into another animal confers passive immunity upon it. In both varieties of serum the potency which may be reached is very remarkable.

Lysogenesis.—As already stated, anti-bacterial sera may have various properties, but for the sake of simplicity we shall consider only one, viz. the *lysogenic*, i.e. the power of producing a solution or destruction of the corresponding bacterium. Pfeiffer was the first to show that if an animal were highly immunized against a particular bacterium (e.g. the cholera spirillum or the typhoid bacillus), and if a number of the bacteria were injected into its peritoneal cavity these underwent *lysogenesis*, i.e. they underwent a granular change, became swollen up and subsequently disappeared. He also showed that a similar result followed if a small quantity of the serum were injected with the bacteria into the peritoneal cavity of a fresh animal. Subsequently arrangements were devised by other observers whereby such phenomena could be produced by the anti-bacterial serum outside the body. Here also, as in the case of toxines, the action of such a serum is within certain limits specific, i.e. is applied only against the organism employed in its production. It should also be kept in view that this power of dissolving organisms is no absolutely new faculty, but may be possessed in small degree by normal serum towards innocuous or much attenuated organisms. In this case, however, there is no trace of specific action.

How then can such a specific dissolving property be explained on Ehrlich's side-chain theory? Theoretically the results with regard to anti-toxines might be directly applied. The action on the bacterial cell might be supposed to be due to the presence of some body in the serum which combines with it and causes disintegration of its structure. (This action is probably analogous to the destruction and disappearance of the nuclei of animal cells, e.g. those of the kidney under the influence of toxines.) Such bodies in the serum

would be used up in the process and would be subsequently regenerated, the regeneration gradually increasing in amount as the process is repeated. While this might theoretically be the case, it is found, however, that the process is a more complicated one than that indicated. Here not a single substance, as in the case of anti-toxines, but two substances are concerned—the action of the serum is a dual one. If such a serum is heated to 58°C . it loses its lysogenic property; it, however, regains it on the addition of a small quantity of serum from a normal animal. If the fresh serum be itself heated to 58°C . before being added it has no effect. There is, therefore, an unstable ferment-like substance present even in normal serum which is essential to the process of lysogenesis. The specific substance, that is the substance specially developed in the process of immunization, is more stable but cannot act alone; it forms as it were a link between the bacterial protoplasm and the more unstable body in normal serum. The specific substance is called by Ehrlich the immune-body (*Immunkörper*), by Bordet the “substance sensibilisatrice.” When agglutination is possessed by an anti-bacterial serum it is retained when the serum is heated to the above temperature. It is still matter of dispute whether or not the agglutinin and the immune-body are one and the same, and this point need not be discussed here. We can say at least, however, that in all probability the agglutinin is a substance formed in the same way and has a combining affinity for the sheaths of the bacteria, producing in them a physical change as Gruber and Durham suppose. As a bacterium is a complicated structure from the chemical point of view, it is quite likely, according to Ehrlich’s law, that more than one anti-substance is formed. In any case the side-chain theory if established will almost certainly apply to the production of agglutinins.

This remarkable lysogenic action, which may be possessed by an anti-bacterial serum, is, however, not restricted to the case of bacteria. The researches of Bordet and of Ehrlich and Morgenroth have shown that similar properties—*caeteris paribus*—can also be acquired towards red corpuscles. It has been known for some time that the blood serum of some animals

has the power of dissolving the red corpuscles of certain other animals. This, however, is not a general rule. But if an animal, whose serum has no dissolving power, is treated with repeated injections of the blood of another animal, this power becomes developed, and may reach a high degree. Bordet showed that the serum of guinea-pigs treated in this way with the blood of the rabbit, acquires powerful haemolytic properties towards the red corpuscles of the rabbit, and the same is true in the case of various other animals. The haemolytic property is, as a rule, exerted only or chiefly towards the blood of the animal employed—that is, within certain limits it is specific. Sometimes the serum acquires agglutinating power towards the same red corpuscles; sometimes this power is absent. Now, the striking fact with regard to this haemolytic process is that its mechanism is practically the same as that of the lysogenesis of bacteria. In the former, just as in the latter, two substances are concerned. One is the specific substance, or, to be more accurate, the substance specially developed in excess—the “immune-body”; it is the more stable, and resists heating at 58° C. The other is a ferment-like substance, the “addiment” of Ehrlich, which is present in normal serum; it is the less stable, being readily destroyed at 58° C. A serum heated to this temperature accordingly loses its haemolytic property, but regains it when a little fresh serum is added. The immune-body thus appears to form a link between the addiment and the red corpuscles—in other words, the conditions of haemolysis are practically the same as those of bacteriolysis.

The relations of the bodies in a haemolytic serum are more easily studied, and this has already been done to a fuller extent than in the case of bacteriolysis. Ehrlich and Morgenroth have proved, for example, that the immune-body enters into direct chemical union with the red corpuscles. This was shown in the following way: A quantity of haemolytic serum sufficient to dissolve a known amount of red corpuscles was taken and heated to 58° C.; the haemolytic function was, of course, thus nullified, the immune-body left in the serum being unable to act alone. This serum was then added to the red

corpuscles, and allowed to act for some time at a suitable temperature. The mixture was then centrifugalized, clear fluid and massed red corpuscles being thus separated. The clear fluid was then tested for the presence of the immune-body, by adding to it a little fresh serum, and thereafter observing its effects on red corpuscles. No haemolysis occurred, therefore the immune-body had disappeared from the fluid. Its presence in the red corpuscles, obtained by centrifugalization, was then tested for by adding to them a little fresh serum, and haemolysis was found to occur at once. In other words, the immune-body had completely combined with the red corpuscles. Other observations, along similar lines, showed that the addiment does not combine directly with the red corpuscles, but does so with the immune-body, though the combination is less firm than that of immune-body with red corpuscles. This process of haemolysis accordingly depends upon the bringing of the ferment-like body of normal serum into relation with red corpuscles by means of the specially developed immune-body. Accordingly, just as in anti-toxine production, the essential element is the production in excess of a definite substance which has a special chemical or combining affinity for the organic substance used in the injections, viz. the red corpuscles. The only difference is, that in lysogenesis the substance produced is also able to fix by another atom-group a ferment-like substance. Ehrlich, as already stated, considers that the normal function of side-chains is to bring molecules into relation to the cell for the purpose of nutrition. He further considers that if the molecule in question be small or comparatively simple, the side-chain merely combines with it; if, however, the molecule be of large size, and requires to be broken up for the purposes of the cell, the side-chain has also the power of fixing a ferment-like substance. The free side-chains constituting anti-toxines belong to the former class; those constituting lysogenic substances or lysines belong to the latter. So far as we know, no complete investigations on the combining relationships of the substances concerned, such as those detailed with regard to haemolysis, have been

carried out in the case of bacteriolysis; but there can be little doubt that corresponding facts will be established here also. The observations on haemolysis have opened up this field of research, and new facts will, no doubt, be quickly established. As an example of this may be mentioned a recently published paper of Metchnikoff, in which he shows that the animal body can acquire the power of dissolving other organized substances—*e.g.* leucocytes—that is, an anti-leucocytic serum may be obtained. And it is therefore quite probable that the lyso-genesis of bacteria, so far from being an isolated phenomenon, will be found to be merely an instance of a general law. It does not seem unjustifiable to anticipate that important practical applications of these and like results will follow.

We have thus endeavoured to show how Ehrlich's side-chain theory has been applied to the various anti-sera. In many respects it is already more than a theory, although confirmation of various points and further investigation are still necessary before it can be considered to be completely established. The leading idea is that in the production of all such sera there is the development of substances which have a special combining affinity for the substance introduced—toxine, bacterium, red corpuscle, etc., as the case may be. The development of these anti-substances represents an over-regeneration and setting free of molecules normally present, which have been lost by combination with the particular substance introduced into the body. We have brought forward this account as the theory is in our opinion the first to afford a rational explanation of the phenomena concerned. To some it may appear to have chiefly a theoretical or speculative interest, but such is not the case. Apart from the importance of attaining a scientific explanation of serum therapeutics and serum diagnosis, and we may add of the spontaneous cure of disease, the knowledge of the processes concerned is of the highest value in the search for further practical results in this department.

BIBLIOGRAPHY.

The following list contains some of the more important papers on this subject. Though by no means complete, it may be found useful for purposes of reference.

Ehrlich.—“Die Wertbemessung des Diphtherieheilserums,” Jena, 1897.

“Ueber die Constitution des Diphtheriegiftes,” *Deutsch. med. Woch.*, 1898, No. 33.

Ehrlich and Morgenroth.—*Berlin. klin. Woch.*, 1899, Nos. 1 and 22.

Weigert, *Deutsch. med. Woch.*, 1896, No. 40. Article, “Antitoxinimmunität” in Lubarsch and Oestertag’s *Ergebnisse der allgemeinen Pathologie*, etc., 1899.

Behring.—*Deutsch. med. Woch.*, 1898, No. 5 ; 1899, No. 1.

Roux and Borrel, *Annal. de l’Inst. Pasteur*, 1898, p. 225.

Bordet, *ibid.*, 1898, p. 688 ; 1899, p. 273.

Metchnikoff, *ibid.*, 1899, p. 737.

C. J. Martin and Cherry, *Proc. Roy. Soc. Lond.*, 1898, p. 420.

Wassermann and Takaki, *Berlin. klin. Woch.*, 1898, No. 4.

Wassermann, *ibid.*, 1898, p. 209.

Pfeiffer and Marx, *Zeitsch. f. Hyg.* vol. 27, p. 272.

Plimmer, *Journ. of Path. and Bacter.*, vol. 5, p. 489.

Cobbett, *ibid.*, vol. 6, p. 193.

TWO CASES OF THROMBOSIS OF THE LATERAL SINUS CONSEQUENT UPON PURULENT INFLAMMATION OF THE MIDDLE EAR—OPENING OF SINUS—LIGATURE OF INTERNAL JUGULAR VEIN—RECOVERY.

BY THOMAS BARR, M.D., AND J. H. NICOLL, M.B.

- I. *Case of purulent thrombosis of left lateral sinus in its whole extent, septic pneumonia of right lung going on to pulmonary abscess and gangrene, consequent upon chronic purulent otitis media. Gradual improvement after clearing out the lateral sinus and ligaturing the internal jugular vein.*

ON the 17th June, 1899, Dr. Barr was called to see David D., aged 30, who had suffered from a discharge from the *left* ear, off and on, for 15 years. The disease was supposed to have been excited by the impact of a snowball. He had for years been in the habit of syringing the ear and insufflating boracic powder. Dr. Barr was informed that for three weeks previously the patient had suffered from pain in the ear so intense as to prevent sleep, that the left side of the head had been swollen for some days, and had been extremely tender to touch, but that this swelling had now disappeared; that there had been constant vomiting for several days with great thirst and loss of appetite; that the chief symptom, however, had been the occurrence for a week past of frequent and most severe rigors, during which, as his sister expressed it, "the bed shook and the teeth rattled." His sister, who had

attended him closely, believed that there had been at least twenty rigors, and that one had lasted 25 minutes, each of them being followed by very profuse perspiration. The temperatures taken by Dr. Alexander of Galston, the family physician, showed typical pyaemic fluctuations, ranging from 100° to 105°, according to the periods of the rigors. Anti-streptococcus serum had been injected twice during the previous week without apparent effect.

On examination the auditory meatus was found filled with granulation tissue, and from the meatus there was a discharge of fetid pus. The pain, which had formerly been worst in the ear, was now felt with greatest intensity in the frontal and occipital regions, while on pressure behind the mastoid area pretty severe pain was elicited. Additional important and ominous symptoms had shown themselves during the previous twenty-four hours, namely, rapid breathing, pain on the right side of the chest, and rusty expectoration. On auscultation crepitation was heard at the back of the *right* lung. Notwithstanding this grave complication, so unfavourable to the prospects of operation, it was thought right to give the patient a chance, and on the day following he was removed by ambulance waggon to the Glasgow Ear Hospital, a distance of twenty miles. Within an hour after his arrival at the hospital Dr. Barr operated on the mastoid. There was no external evidence of mastoid mischief, but, on making a long incision and reflecting the auricle and membrano-cartilaginous meatus well forward, the greater part of the postero-superior wall of the osseous meatus was found to be destroyed by caries; from this sprouted the granulation tissue which filled the meatus. On further removal of bone a large cavity, *packed with cholesteatomata*, was found to extend back to the posterior fossa of the cranium, and the cholesteatomata covered the sigmoid part of the lateral sinus, which was exposed by the disease over a considerable part of its extent. The cholesteatomata, with cario-necrotic debris and granulation tissue, were thoroughly removed; the sinus and dura mater were normal so far as colour, appearance, and touch were concerned.

For that and other reasons it was not considered desirable to open the sinus, but to wait and see the effect of this operation. The large opening was therefore simply treated by iodoform packing.

For the next eleven days, while only one rigor was observed, the temperature had wide ranges extending from 97° to 106° . The respirations were rapid relatively to the pulse, ranging from 28 to 36 in the minute, and the pulmonary condition developed with consolidation. The question during these days was, were these pyaemic temperatures connected with pus formation in the lungs, or were they due to continued septic infection from the sinus? Dr. J. H. Nicoll was asked by Dr. Barr to see the patient, and the question of ligaturing the internal jugular vein was seriously considered. Dr. Finlayson was also consulted (although he did not at this time see the case), but he was rather unfavourable to the proposal for ligaturing the vein in the presence of such a condition of the lung. However, the pyaemic temperatures continuing, it was decided that the internal jugular should be tied and the sinus freely opened, so as to avoid, if possible, any further infection.

On the 29th June, eleven days after the previous operation, Dr. Nicoll applied two ligatures to the left jugular at the level of the cricoid cartilage. The vein, which was left undivided between the ligatures, seemed collapsed and empty. Immediately afterwards Dr. Nicoll opened the sinus, which was found occupied by softened purulent thrombi, and there was no blood stream. The bone over the lateral sinus was removed as far as to the Torcular to the extent of an inch in breadth—a large amount of bone being in this way removed (see Fig. I.). The sinus, which was somewhat rough on its outer surface, was opened, and in almost its whole extent, from the jugular foramen to the Torcular, was found occupied by pus and thrombi. The patient bore these formidable operations well, and distinct improvement soon followed, as shown by lower temperatures and much less violent fluctuations. The condition of the right lung was the main source of anxiety; there was now purulent expectoration, but we were encouraged by the fact that Dr. Finlayson, who saw the

patient at this stage, expressed a favourable opinion of the prospects.

On the 16th July, a fresh complication was observed, in the form of a large abscess over the sacrum—where no pain had been complained of—the swelling being first observed by Dr. Finlayson while examining the back of the lungs. On the same evening a large quantity of matter was evacuated from this abscess, and it did not give much further trouble.

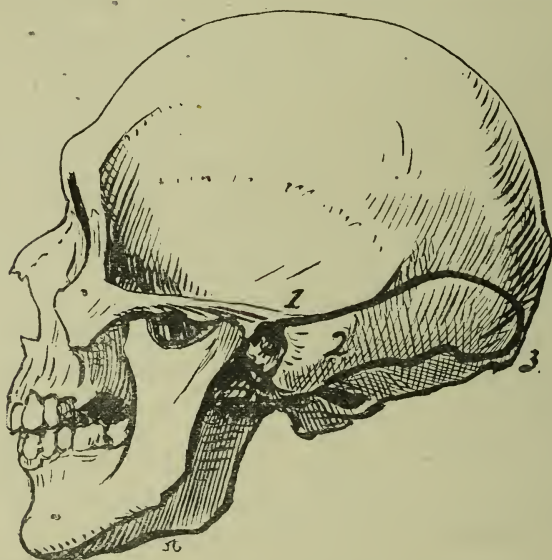


FIG. I.—Shows area of bone removed in order to expose the lateral sinus: (1) above auditory meatus, (2) over posterior border of mastoid, (3) opposite situation of Torcular.

At the end of July, a fortnight after the evacuation of the sacral abscess, the breathing somewhat suddenly became much more embarrassed, with distressing cough and pain in the right side of the chest, while the purulent expectoration became very profuse, to the extent of a cupful in the course of a night. The pulmonary distress continued, and the condition at the beginning of August resembled the advanced stage of phthisis pulmonalis—rapid breathing, extreme emaciation, hollow cheeks, great paroxysms of coughing with copious purulent expectoration, also high temperature and profuse

perspiration, but no rigors. In so far as the cranial condition was concerned everything seemed satisfactory—the wound in the scalp had been sutured and was now quite healed; we were confident that there could be no fresh infection from the sinus or ear. It was clear, however, that there were purulent foci in the right lung, resulting from septic infarctions. The left lung, however, still remained apparently unaffected, and we hoped that his strength might enable him to survive the terrible complication in his right lung. At this time Dr. John Rowan made an ophthalmoscopic examination; and found the “optic discs somewhat pale on the temporal sides and edges slightly indistinct, veins slightly full, fundi practically normal.”

During the early part of the month of August he remained in somewhat the same condition, with the exception that the purulent expectoration, which continued copious, now emitted a gangrenous odour, especially when the patient lay on the left side; a whiff of air having a gangrenous odour was perceived during coughing. Yet as the month went on his general condition seemed to become no worse, but rather better. The temperature ranged from 99° to 102° , and the strength seemed to improve, with some diminution, though not great, in the purulent expectoration. The latter was examined bacteriologically and found to contain numerous chains of streptococci, but no tubercle bacilli. Towards the end of August the improvement seemed to be such as to justify his removal to his home in the country, partly with the hope that the change to the country air might be beneficial. He was removed on the 30th day of August, still expectorating pus, though in less quantity, but gaining in strength. The ear was entirely dry and the extensive incisions were completely healed over, the large gap in the bone was also filled with firm material. The temperature for a week before leaving the hospital ranged from normal in the morning to 100° in the evening.

The change to the country was beneficial, and he went on improving. The temperatures continued to be normal in the mornings and from 99° to 100° in the evenings. On the 14th

October Dr. Alexander sent the following notes: "David D. is now walking about daily, but is rather breathless on any exertion. He has practically no cough except in the mornings, the expectoration has diminished gradually, and now there is only a little nummular spit in the mornings, and this is entirely devoid of any gangrenous odour. He is now putting on flesh rapidly, and I should think that he must have increased to the extent of at least a stone. The temperature has only been taken in the morning, when it was normal. The physical signs over the left lung are quite normal, except that the respiratory murmur is rather puerile. Over the front of the right side there is no dulness, but on deep inspiration and on coughing there are still some crackling rales; over the back the rales are also present on deep inspiration, but *in the lower half of the right lung there is a large cavity* with loud rales. The rales all over the right side are diminishing gradually, and the lung is evidently healing up."

12th November, 1899.—Dr. Finlayson examined patient to-day and found no definite signs of cavity in the right lung, only dulness at certain places with some moist rales, also a want of due expansion on right side in front. Patient reports that there is scarcely any cough or expectoration. The most striking change is the notable increase of flesh. Instead of the emaciated phthisical look, he is quite full in the face and plump in the body. He thinks he has gained about two stones since the early part of his illness. There is a deep groove in the scalp extending from the mastoid to the external occipital protuberance, and the long gap in the bone seems to be converted into dense fibrous tissue (see Fig. II.). There is an aperture still remaining behind the auricle. This leads into a dry cavity which represents excavated mastoid, tympanum, and meatus. The purulent process in the ear seems to be entirely at an end. The temperature still rises somewhat in the evening, though normal in the morning. There is also some shortness of breath during exertion.

Observations.—The following points are worthy of emphasizing:—1. The serious significance of rigors, high temperature, pain behind the mastoid, sickness and vomiting in connection

with chronic purulent inflammation of the middle ear. 2. The vital importance in such cases of prompt and thorough removal of all sources of septic infection. 3. If the sinus be not opened at the time of the mastoid operation, this should not be long delayed, in the event of rigors or high temperature recurring afterwards; and *we must not hesitate in doing this, although*



FIG. II.—Photograph of patient's head, taken 4½ months after operation, showing depressed cutaneous scar over fibrous tissue occupying gap in skull left by removal of bone (indicated in Fig. 1.). Behind the auricle there is a dry cavity remaining after the mastoid operation.

the outer surface of the sinus appears normal. 4. This case shows that, even when there is evidence of pulmonary mischief, such operations as ligature of the jugular and opening of the sinus may be safely and with advantage carried out. There is no doubt that distinct amelioration took place after these operations, and the patient owes his recovery, as far as that has taken place, to these operations in connection with the

preceding mastoid one. 5. While the ligature of the internal jugular vein may, by some, be still regarded as a debatable procedure in such cases, it is a wise precaution, as it diminishes the chance of purulent debris making its way into the circulation. Then the operation is the more justifiable as it is by no means a dangerous one in the hands of an experienced surgeon, and in thrombosed conditions of the lateral sinus.

II. Case of extensive organized thrombus in right lateral sinus, connected with acute purulent otitis media and extra-dural suppuration. Ligature of internal jugular vein, opening of sinus, and removal of part of thrombus. Recovery.

J. L., a gentleman aged 53, having a very healthy physique, consulted Dr. Barr on 20th May, 1899, owing to acute pain and throbbing in the right ear, of a few days' duration. The pain had come on just before a railway journey to London, and it was very severe while in London. Dr. Barr found acute otitis media, without, so far, any perforation. There was marked dulness of hearing; a watch, heard ordinarily at a distance of forty inches, was heard only one inch off. The left ear was also dull, and there were in the meatus of this ear several small exostotic growths.

The pain continued more or less for a fortnight, when profuse discharge appeared, with relief to the pain. Soon afterwards, however, the patient was seized, in Glasgow, with a severe rigor, and with difficulty he reached a friend's office, where the rigor continued for a considerable time. This was followed, when he got up to walk, by giddiness and staggering. On the same day he travelled home to Helensburgh, feeling in a dazed condition, and had to be taken to his house in a cab.

Soon afterwards pain was complained of behind the ear, and on visiting him at Helensburgh on 15th June, Dr. Barr found redness and swelling over the mastoid, which was also

painful on pressure. Under chloroform a free incision was made down to the bone ("Wilde's incision"). No pus appeared at the time, but in a few days matter began to come from the opening, while the discharge from the meatus ceased. The discharge from the wound continued, with the formation of granulation tissue, while the edges of the incision became much swollen and thickened. There was also a good deal of throbbing, and at times he seemed to have pain on pressure behind the mastoid in the region of the mastoid vein. There was, however, little or no elevation of temperature. The patient came up to the Glasgow Ear Hospital on 3rd August, and the mastoid surface was freely exposed. A very small orifice was found penetrating the cortex, just behind the bony meatus, lower down than the antrum, this orifice being evidently the source of the discharge. It was enlarged with the rotating burr and gauge, and a cavity of considerable size freely opened out. From this cavity granulation tissue and pus, with cario-necrotic debris, were thoroughly removed, and its walls were dusted with iodoform and boric acid, followed by packing with iodoform gauze. The meatus was evidently dry, and this had been the case for a considerable time. The gauze still continued to be saturated with pus at every dressing, although little or no pain was complained of. On 31st August, the patient being again placed under chloroform, the cavity was carefully explored, but no special source of the pus could be discovered beyond firm granulation tissue. The soft tissues over the bone were, however, still remarkably swollen.

The discharge persisted to the extent of soaking the dressings, and about a fortnight afterwards the patient had a distinct sensation of creeping cold over the body, not, however, amounting to a rigor. At this stage Dr. J. H. Nicoll first saw the case, and it was decided to make a further exploration in search of a source of the pus; this was done on the 18th September. Immediately before this operation the patient had another chilly feeling. A small orifice was at last found at the bottom of the cavity, leading to a fine canal extending somewhat deeply. More bone was removed and the canal

opened out as thoroughly as possible. It seemed to terminate in a pouch, and we supposed that the root of the matter had at last been reached. Three days afterwards a slight rigor was experienced, with elevation of temperature to 102° , the highest observed during the illness.

Pus still continued to soak the dressings, and on the 25th September the patient was again put under chloroform, and Dr. Nicoll removed the bone from over the sigmoid sinus and neighbouring dura mater to a considerable extent. The sinus presented a rather remarkable appearance, the whole sigmoid part with a portion of the horizontal being converted into a hard, round mass, covered with firm granulation tissue. The latter was carefully curetted away and a wedge of solid material (sinus wall and contained thrombus) was removed and examined by Dr. Teacher, who reported in the following terms: "In my opinion the structure is a mass of granulation tissue, for the most part in advanced stages of its conversion into fibrous tissue (practically scar tissue) which has replaced a thrombus. It must be a formation of considerable age." The source of the pus seemed to be the lower part of the sinus, and this was thoroughly exposed, the whole cavity being packed with iodoform gauze. From this onwards the case progressed favourably, the purulent discharge at length ceased, and the cavity became lined with healthy granulation tissue.

The propriety of ligaturing the internal jugular vein was now considered, and it was decided, in order to avoid any risk of systemic infection from possible purulent disintegration, especially at the lower part of the thrombus, to ligature the internal jugular vein, which was done by Dr. Nicoll on the 19th October. The patient after this made an uninterrupted recovery. The wound in the neck healed by first intention, and the cavity behind the ear is rapidly filling up, the interior of the ear being quite dry.

Observations.—1. The sequence of events in this case was probably the following:—Otitis media purulenta followed by abscess in the vertical portion of the mastoid, then by extradural or rather extra-sinus abscess, with thrombosis of the sinus and organization of its contents into connective tissue.

2. A feature of this case as contrasted with the previous one was the comparative absence of rigors and high temperature. Only one well marked rigor occurred and other three slight ones, while the temperature only on one occasion reached 102° , and was nearly always under 100° .

3. This comparative absence of rigors and high temperature is probably accounted for by the effective closure of the sinus with firm thrombus, which, instead of undergoing purulent disintegration with systemic infection, became firmly organized into connective tissue.

4. It is probable that in an acute case purulent disintegration of the thrombus is less likely to take place than in a chronic case. Unfortunately there was no bacteriological examination of the inflammatory products, so that the nature of the micro-organism associated with this condition was not determined.

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